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GUY'S HOSPITAL REPORTS.

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N. DAVIES-COLLEY, M.A., M.C.,
AND
W. HALE WHITE, M.D.

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CONTENTS.

PAGE

I. In Memoriam:
Sir William Gull xxv

II. Report of Cases of Anthrax or Malignant Pustule under the care of Mr. Davies-Colley; with Notes upon the Treatment of this Affection by the External and Internal Use of Ipecacuanha. By N. Davies-Colley . . . 1

III. Sarcoma of the Tongue. By J. H. Targett, M.S. . 21

IV. An Analysis of the Cases of Chorea which have occurred in the Hospital during Eleven Consecutive Years. By E. W. Goodall, M.D. . 35

V. A Case of Fracture of the Coracoid Process uncomplicated with other Injury, with a Study of the Lesion. By R. Clement Lucas, B.S. . 45

VI. On Primary Malignant Disease of the Liver. By W. Hale White, M.D. . . . 59

VII. The Pathology of Acute Infective Periostitis and Acute Necrosis of Growing Bone. Being the Report of an Investigation carried on in the Workrooms of the Royal College of Surgeons of England and the Pathological Institute at Leipzig. By A. H. Tubby, M.S. . . 77

VIII. Chiefly concerning Bruits. A Lecture on Pathology from a Course upon "Points in the Pathology of Diseases of the Heart." By James F. Goodhart, M.D. 101

IX. A Case of Glanders, with the Results of Cultivation and Inoculation Experiments. By J. W. Washbourn, M.D. . . . 127

PAGE

X. On the Pathology and Prognosis of Pernicious Anæmia. By W. Hale White, M.D. . 149

XI. Experiments on some Antiseptics and Disinfectants. By Percy C. Evans, M.B. (Communicated by J. W. Washbourn, M.D.) . . 195

XII. Congenital Wry-Neck (Caput obstipum congenitale; Torticollis congenitalis); with Remarks on Facial Hemiatrophy. By C. H. Golding-Bird . 253

XIII. Note on the Urine in a Case of Phosphorus Poisoning. By Ernest H. Starling, M.D., and F. Gowland Hopkins, B.Sc. . . 275

XIV. A Case of Compression Paraplegia. By E. W. Goodall, M.D. With an Account of the Microscopical Examination of the Cord. By E. Goodall, M.D. 279

XV. Two Doubtful Cases of Tuberculosis, in which the Diagnosis was cleared up by Inoculation. By J. W. Washbourn, M.D., and W. H. A. Jacobson, M.Ch. 293

XVI. Abormalities observed in the Dissecting Room of Guy's Hospital during the Sessions 1888—1890. By L. H. Dunn, M.S., J. W. Washbourn, M.D., and J. H. Targett, M.S. . . . 299

XVII. A Case of Internal Derangement of the Knee-Joint, from Splitting of the Internal Semilunar Cartilage, Treated by Operation. By N. Davies-Colley 317

XVIII. List of Specimens added to the Pathological Museum during the year 1888-89. By G. Newton Pitt, M.D., and Lauriston E. Shaw, M.D. . 325

List of Pupils who have passed the Examinations of the several Universities, Colleges, &c., 1889 . 349

List of Prizemen and Medallists, 1888-89 . 355

List of Pupils who have received Appointments at Guy's Hospital, 1889 . . . 356

LIST OF ILLUSTRATIONS.

PLATES.

TO FACE PAGE.

Autotype portrait of Sir William Gull . . . xxv

Mr. Tubby.

Plate illustrating his paper on Acute Infectious Periostitis and Acute Necrosis of Growing Bone . 100

Dr. J. W. Washbourn.

Plate illustrating his paper on a Case of Glanders . 148

ZINCOTYPES.

PAGE

Mr. Dunn, Dr. Washbourn, and Mr. Targett.

Seven Zincotypes, illustrating their paper on Abnormaltities observed in the Dissecting Room of Guy's Hospital . . . . 305—314

Mr. Davies-Colley.

One Zincotype, illustrating his case of Internal Derangement of the Knee-Joint . . . 318

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John Hopkins Museum (care of Mr. Hurd).
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Journal de Thérapeutique (care of M. le Dr. Labbée, 10, Rue de Turbigo, Paris).
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Yours very truly
William Rockhill

In Memoriam.

SIR WILLIAM GULL.

The late Sir William Gull was born on the last day of the year 1816, at Thorpe-le-Soken, a secluded Essex village near Walton-on-the-Naze.

The accident which determined his whole future life was his being born and bred on the Essex property of Thomas Guy's foundation. In recent years these estates have become the least valuable of any belonging to the Hospital, but in William Gull they produced a return to make up for many barren years.

He was still a child when his father died, and the family was left in straitened circumstances. He appears to have owed his character and training to his mother and his early education to himself.[1]

The late Mr. Benjamin Harrison, who was for fifty years the despotic Treasurer, the sagacious and beneficent ruler of the Hospital and School, was struck by the appearance and the intelligence of the youth whom he found teaching in the village school of Thorpe. He offered to give him work as a clerk in the counting-house at Guy's Hospital, and when he came up to enter on his new duties told him, "If you help yourself, I shall be able to help you." The bargain

[1] It has been repeatedly stated that Gull was an Old Blue, but this was contradicted after his death by his only surviving sister in a letter to the 'Times,' and on inquiry at Christ's Hospital I find that his name does not appear upon the books of the school.

was well kept on both sides. Gull's first duty was to copy out in fair hand the catalogue of the Guy's Museum which had been drawn up by that eminent pathologist and excellent man, the late Dr. Thomas Hodgkin. Meantime he worked on at Greek and Latin and mathematics, and in 1838 was able, with this self-tuition, to matriculate at the then new University of London. Mr. Harrison, faithful to his promise, thereupon offered to procure him a free entry to the Guy's School of Medicine, and for this purpose, at a meeting of the staff, proposed that William Gull should be made a perpetual student. This was agreed to, and thus his wonderfully successful career in medicine was begun.[1]

He took his M.B. degree in 1841 along with the brilliant and justly popular Edmund Parkes, from University College. Dr. Quain graduated the year before, and Sir Alfred Garrod and Dr. George Johnson the year after. Gull took honours in medicine, in surgery, and in physiology. In 1846 he proceeded to the M.D. degree, and was the first of the many gold medallists who have come from Guy's school. Some time before, he had been appointed by his friend, the Treasurer, to a resident office in the Hospital, which gave him for about nine years almost unequalled opportunities of daily clinical observation.[2] He visited the wards once and often twice every day of the week, and, living in one of the Hospital houses in King Street (since pulled down), he heard and saw everything that went on. Mr. Stocker, who not long before had succeeded his father as Resident Apothecary to the Hospital, was Gull's companion and friend; and his good judgment of character, with the influence that his

[1] It is only just to add that during his studentship, and the still more trying period between taking his degree in 1841, and being appointed assistant physician, he was treated with the utmost kindness and consideration by the medical staff. He always spoke with special admiration and gratitude of the younger Dr. Babington (Benjamin Guy, who married Dr. Bright's sister, and only died in 1866) and others of his teachers and subsequent colleagues. The good sense and good taste on the one side and the liberal cordiality on the other which marked his early relations with them do honour to both.

[2] Few now survive who remember the period between 1840 and 1850, and I am not sure as to the exact time during which Gull held this resident appointment. I have heard it stated as fifteen years, but that must, I think, include his period of studentship (1837 to 1841).

enthusiastic devotion to the honour and glory of Guy's commanded, were often of service to his gifted comrade. He himself has again and again spoken of the invaluable opportunity he then enjoyed of becoming acquainted with the aspect and phases of disease. His duties seem to have been multifarious and perhaps undefined. He was Medical Tutor, he had special charge of the lunatic patients (who then lived in the present Clinical Building), and he was also associated with Mr. Stocker in his care of the wards during the absence of the staff—for this was many years before the first House Physician was appointed.[1]

To these various duties was added, in 1843, the Lectureship on Natural Philosophy, in accordance with the old rule that a physician should be competent to teach physics as well as physic. An eminent German professor, visiting England some years ago, was astonished to find, as he put it, that every professor of internal pathology in this strange country begins by teaching botany. The elder Dr. Babington, for example, like Boerhaave at Leyden, lectured on chemistry, on materia medica, on botany, and on medicine. There is, however, no doubt that a good acquaintance with these preliminary scientific subjects is the best preparation for the scientific work of a physician. Dr. Golding Bird, who preceded Dr. Gull in this lectureship on physics, was so excellent a teacher that his 'Manual of Natural Philosophy,' published for the use of his class, became the most widely and justly appreciated introduction to the subject until the appearance of Ganot's 'Physics.'

It is recorded that even in King Street Dr. Gull began to acquire considerable practice outside the Hospital; and when in 1848 he married and soon after moved to Finsbury Square,

[1] The house surgeon held office for at least six months, and often for as much as two years together. The office of clinical assistants (or, as they were then called, clinical clerks, in distinction from the ordinary ward clerks) had been only lately instituted (1836). The dresserships were not given after a competition as they now are, but as the result of paying heavy fees to the surgeons, a source of emolument which was afterwards very liberally surrendered by the surgical staff at Guy's and at the other great hospitals. Those who lived in the full glory of the old Guy's dresserships would not condescend to minor operations which house surgeons in other hospitals were only too glad to be occasionally allowed to perform.

he rapidly became popular as a physician. He succeeded the late Dr. Jefferson in the house No. 8, on the west side of the square. He and his neighbour Dr. Oldham used to walk up the City Road for exercise before breakfast, sometimes accompanied by another life-long friend, Dr. Hermann Weber.

Dr. Gull was appointed Assistant Physician to Guy's Hospital in 1851, and was Lecturer on Physiology in the Medical School for several years (1846—1856). He was also Professor of the same subject at the Royal Institution (1847—1849), where it is said that he accomplished with unusual success the difficult task of lecturing on a technical subject to an uninstructed audience. In 1858 he became full physician on the death of Dr. Hughes, and lectured on Medicine in conjunction with Dr. Owen Rees, who died only a few months before his colleague, and from the same disease.

Dr. Gull's practice had steadily and rapidly increased during his residence in the City, and advanced at a still quicker rate when he removed to 74, Brook Street, on the death of Dr. Todd, in 1862. His successful management of the severe attack of enteric fever from which the Prince of Wales suffered in 1871 made him still more widely known. The Queen acknowledged his services by making him a baronet, and appointing him one of the physicians to the Crown, and the Prince always showed a generous and grateful sense of what he owed to his physician's skill.

Few men have been so successful in practice or have left so large a professional fortune. His striking presence, his searching scrutiny, his minute and deliberate examination of every case, and the few carefully and slowly uttered words in which he delivered his judgment,[1] sometimes with epigrammatic pungency, often with encouragement, and never without sympathy—all combined to give him an almost unequalled ascendency over his patients. His manner was his own, and sprang naturally from the habit of his mind. It was just the same in a hospital ward as in a palace, and the poorest of his patients leant on his oracular statements, some-

[1] Καὶ κίνησις δὲ βραδεῖα τοῦ μεγαλοψύχου δοκεῖ εἶναι καὶ φωνὴ βαρεῖα καὶ λέξις στάσιμος ('Arist. Eth. Nich.,' iv, 7, 34).

times with hope and sometimes with resignation, but always with comfort; while the richest were taught to restrain loquacity, to answer truthfully, and to follow out directions implicitly.

In consultation he was not so uniformly happy. Where he met with his equals, like Sir William Jenner or Sir James Paget, or his juniors, like his own pupils, he was most courteous and helpful. But in cases where he felt his superiority, and where it was not equally felt on the other side, his masterful manner and justly founded self-confidence sometimes gave offence, which he was far from intending, and heartily regretted. This independence of judgment and confidence in his own actions as well as motives led to the two or three occasions on which he incurred some degree of professional censure. It is pleasant to record that a distinguished graduate of his University, who believed himself to have been slighted, was one of Sir William Gull's supporters when he was spoken of as the future President of the College of Physicians, and a kind and welcome visitor during the time that he was laid aside by illness. Another eminent physician who had called at Gull's house one evening on a matter of college business, and had found him in great indignation at what he thought unfair treatment, left with the wounded feelings of an unsuccessful peacemaker. But before he had gone to bed an express messenger arrived with a letter in which Gull warmly acknowledged his services, regretted his own warmth, and effectually dispelled every feeling of annoyance.

The defects of his great qualities seem to have prevented his excelling as an administrator on committees or in conjoint work. Nevertheless he gave both time and thought to what he considered public claims. His judgment was sound. He spoke little and to the purpose, and when occasion arose for a prolonged address, his admirable delivery and happy choice of words gave distinction to opinions which were always broad, liberal, and sagacious. He was a valued and useful member of the Council of the Royal Society, and of the Senate of the University of London, on which he was the first medical graduate appointed; and as chairman or orator, his address as President of the Clinical Society, his address

in Medicine before the British Medical Association, and his Harveian Oration before the College of Physicians were conspicuous for their ability and influence.

In the hospital wards Dr. Gull was admirable both as a physician and a teacher. He was punctual, methodical, and most considerate for his patients. What he most insisted upon and practised was thorough, systematic investigation of every organ. He had a keen and well-practised eye for slight differences of expression, of complexion, and of attitude. No peculiarity in the speech or the gestures of a patient escaped him. He would patiently and critically search for fever spots, for the runs of the acarus, or for gall-stones in fæces. He was an excellent auscultator; and although the inventions of the ophthalmoscope and the laryngoscope came too late for him to use, he appreciated their value, and gratefully accepted the information derived from them which he could learn from younger men.

Being asked by a colleague to give his opinion on a difficult case of obscure cerebral disease with marasmus, he put his hand under the bedclothes as if to feel the abdomen, and then said, "Give him iodide of potassium." He had discovered a syphilitic sarcocele.

This minute and thorough examination of the patient which we owe most of all to the great French clinical school of the second quarter of the century has happily long ago been taught and practised in Paris and London, in Dublin and Edinburgh, in Leipsic and Vienna, in Boston and Philadelphia. It was the method successfully taught and practised by the late Professor Hughes Bennett, of Edinburgh, who learnt it in Paris, and by his eminent pupil, the late Dr. Murchison. The merit of Gull, as of Stokes, Oppolzer, Trousseau, and other great physicians, was that he added to this methodical investigation of facts the insight and judgment without which a man may be an admirable clinical teacher and yet entirely fail in the diagnosis of really difficult cases, in the estimation of probabilities, and in wise therapeutics. For practical medicine is an art; and when all the facts which the most elaborate examination of the patient's actual state can supply have been brought in due order before us, and the seat and nature of the anatomical lesion has been accurately determined, there

still remains the due appreciation of its significance in that particular case, of the probabilities of its future progress, and of the most suitable methods of meeting it. Indeed, in many cases the result of physical diagnosis is little more than negative, and we have to depend entirely upon the same half-unconscious use of experience in combination with slight and almost indescribable indications which is depended upon in critical moments by the physician no less than by the engineer, the seaman, the statesman, and the general.

While not inferior to the best modern teachers in accuracy and thoroughness of searching for physical signs of disease, Gull added to this the vision and the faculty which depends upon long and well-used experience combined with a certain happy mixture of boldness and caution like that which decides a doubtful field of battle.

He was called in to see a young man in a condition of hysterical mania, and was assured that he had had brain fever and was delirious for several days, in proof whereof broken balusters were shown him. "No," he said, "that is not brain fever. Break some more balusters if you like, you will get quite well.—He could not have had fever for several days," he remarked as he came away, "or he would have lost flesh."

In visiting a case of acute pneumonia in a head master of a public school he noticed that though the weather was cold there was no fire in the room and the patient's hands were cold, that he was sleepless, and, notwithstanding his anxious depreciation of his symptoms, much alarmed as to his state. "Sir," said he, "our friend here has got you through the worst of your illness; you will get quite well;" and, checking an effusive speech from the half-hysterical wife with the repeated remark, "Ma'am, your husband will get quite well," he ordered a fire in the room and a grain of opium that evening. The patient was made comfortable, had a good night's rest, and recovered well.

It ought in fairness to be said that those who, like the writer, learned their first lessons in clinical observation from Gull, had a higher opinion of his powers than those somewhat senior, who could compare him with his more brilliant colleague Addison. While probably inferior in perseverance, in knowledge, and in therapeutical skill to Gull, Addison was

no doubt his superior in original force and vigour of intellect, in natural genius for discovery, and in power of speech. His striking person, his eloquent tongue, his frank and masterful manner, and even the hasty temper and outspoken contempt for everything and everybody that he disliked, made him the idol of his pupils, who seem scarcely to have done justice to the less demonstrative, more patient and self-restrained manner of the younger physician.

As a lecturer Gull was careful, instructive, and interesting, full of impressive aphorisms and ripe conclusions, using apt and striking metaphors, but only sparingly, and enforcing what he taught by a dignified, slow, and careful reiteration, which never wearied, and which it required more than average carelessness to forget. His lectures on Medicine were as accurate, finished, and full of information as those of Dr. Alfred Swayne Taylor upon Chemistry, but they lacked of course the additional interest of the admirably planned and always successful experiments of the latter course. They were often compared by us students with the surgical lectures of Mr. Hilton, and each had his partial admirers; but although that eminent surgeon had no grace of manner or elocution, and not much dignity of style, yet the native shrewdness of his remarks, the admirable way in which he told the story of a case, the pungent homeliness of his illustrations, his unrivalled application of anatomical knowledge to illustrate his subject, and the sterling good sense and good humour which pervaded the whole, made his teaching in the wards and his lectures in the theatre more original, more impressive, and therefore more useful than the clinical teaching and the systematic lectures of his medical colleague.

It would be a great mistake to suppose that Gull's excellence as a practical physician was limited to power of diagnosis, appreciation of the patient and his conditions, or facility in acquiring and retaining that confidence of the sick man which in most cases is essential for carrying out treatment, and in many is itself a help to cure. He was generally supposed to be a sceptic as to therapeutics, and his treatment to be what some people call "expectant" and others "do nothing." As in most cases of general reputation, the conclusion, so far as it was incorrect, was in great measure due to the very man

whom it wronged. If a physician honestly believes that medicine is powerless over disease, he need not relinquish his practice, for it is often well worth a patient's while to ascertain whether he is really ill or not, and what is the nature of his complaint; he may often wisely pay for the knowledge of what is likely to happen, and what cannot be cured may often be prevented. But although negative practice might still be followed for the sake of diagnosis and prognosis and prophylaxis, yet an honest disbeliever in therapeutics could never prescribe a potion or a pill without losing his self-respect. As a matter of fact, Sir William Gull often prescribed no drugs whatever, and his prescriptions when he wrote them were of extreme simplicity : ten or fifteen drops of tincture of steel three times a day, sulphate of magnesia and carbonate of soda, mineral acids in infusion of gentian or orange, aloes and compound rhubarb pills—these were his frequently recurring formulæ. To these, in suitable cases, were added quinine, digitalis, opium, arsenic and mercury, in moderate but efficient doses. He was never tired of exposing the absurdity of much of the traditional polypharmacy. He would show how much harm may be done by the vigorous treating of half-understood diseases, and he once said that if every drug in the world were abolished a physician would still be a useful member of society. To appreciate his position, we must remember something of the unquestioning faith in bleeding and blistering, purging and physicking, which was still held when Gull was a student. The physicians of the first half of the present century seemed to have no conception that many diseases naturally tended to recovery if not interfered with, and that others as naturally tended to death, however treated. Their erroneous notions of physiology and pathology, their not yet exploded remnants of mediæval superstition, their almost unsuspected ignorance of the processes and even the detection of disease, and their boundless credulity in the action of remedies, led to just criticism in the minds of the more thoughtful of the laity, and to something like negation among a few in the ranks of the profession who were not bound by the fetters of tradition—such as Sir John Forbes and Dr. Hughes Bennett, of Edinburgh. The astonishing fact that under treatment by Hahnemann's

sect patients did not always die, led thoughtful men to open their eyes.

Gull's treatment of fever and of acute rheumatism were valuable contributions to the scientific study of therapeutics; and he once said to the present writer after his retirement from practice, "One thing I am thankful that Jenner and I have together succeeded in doing. We have disabused the public of the belief that doctoring consists in drenching them with nauseous drugs." Nevertheless, those who knew Gull's practice, either in the Hospital or in private, are well aware that his scepticism was perfectly reasonable, and his therapeutical faith all the stronger because it was discriminating. He would give mercury in syphilitic cases with the utmost confidence; he purged and used steel and quinine and arsenic just as freely as other physicians; and in cases of cardiac dropsy his belief in "Addison's pill," as he always called it, of digitalis, squills, and mercury, was unbounded. He used opium also with rare skill and judgment.

He often did not do himself justice. His paper on the treatment of rheumatic fever was headed "Cases of Acute Rheumatism treated by Mint-water." But really his treatment of this disorder was most thorough and efficient. He had the patient's bed screened from the rest of the ward, his friends were not allowed to visit him, he was not suffered to be moved, and no examination of the heart was made beyond what could be carried out without disturbing him. He was put on low diet with plenty of barley-water to drink, the inflamed joints were carefully wrapped in cotton wool and protected with cradles, and he always had enough opium at night to secure sleep and prevent painful startings of the limbs.

Having seen this treatment carried out at the same time and in the same hospital with that by lemon-juice, that by alkalies, and the old-fashioned eclectic treatment of a little purging, a little mercury, a few leeches, a little colchicum, a little nitre, and then again a purge—the present writer has no hesitation in saying that, while the last method was ineffectual or mischievous, the treatment by lemon-juice sometimes efficacious but often useless and occasionally dangerous, and that by alkalies often efficient but often failing and always tedious, Gull's expectant (or, as I should call it, rational and

physiological) treatment of rheumatic fever was by far the most effectual in relieving pain, shortening the disease, and securing a good convalescence.

That the method has since been superseded by the discovery of the remarkable power of salicylic compounds upon this disease was acknowledged by Gull among the rest.

The popular belief that he did not believe in medicines did great harm, and did him injustice—injustice which, as usual, was not quite undeserved by the sufferer. But there are not wanting signs of a new era of meddlesome medication and reckless polypharmacy. The generation which is now getting old was brought up under the healthy reaction from the old tyranny of drugs; but the new generation is familiar with scores of new, much vaunted, but little tested remedies, most of them uncertain, and many dangerous in their effects. The practice of medicine in Germany, and to some extent in America, is with many honorable exceptions rash, meddlesome, and sometimes mischievous, repeating the old blunder of assuming that every disease has its appropriate remedy, and too often treating each symptom as it arises with the self-confidence and ignorance of a homœopathist.

The worst of the almost daily introduction of highly vaunted remedies, to be used, abandoned, and replaced by others destined to live as short a life, is to produce in scientific tempers complete scepticism in therapeutics; and the worst of the universal scepticism which clever men are apt to feel or to affect is to encourage relapse into pharmaceutical credulity.

We have spoken of Sir William Gull as an accomplished and wonderfully successful practitioner; we have tried to do justice to his remarkable talents as a clinical teacher. But a third branch of activity, without which no success in practice gives reputation worth having, is the advancement of professional knowledge.

In this respect Gull's services have perhaps scarcely been sufficiently appreciated, for he wrote little, and only in scattered papers in these 'Reports' or elsewhere. He never published a book, and his separate contributions were never united as they might have been in a single octavo volume.

But they were important enough to entitle him to rank with such of his contemporaries as Jenner and Gairdner.

His first published paper was one in conjunction with Dr. Addison on that remarkable affection of the skin to which they gave the cumbrous name *Vitiligoidea plana et tuberosa*. It has since received the now familiar title Xanthelasma. The paper referred to described all its essential features and figured its two principal varieties; and with the exception of a mere line describing a drawing in Rayner's 'Atlas' of the commonest form, which he called "*plaques jaunâtres des paupières*," this paper was the first account of the singular disease it describes. It appeared in the 7th and 8th volumes of the second series of these 'Reports,' pp. 265, 149.

His second paper was a therapeutical one on the value of electricity as a remedial agent (1862, vol. viii, p. 81), and in the same volume there is an anatomical account of a parthenogenetic tumour attached to the heart of a sheep (ibid., p. 145).

He contributed in 1866 a valuable paper on the curious condition now known as intermittent hæmoglobinuria, and to the 11th and 12th volumes his well-known report, published in conjunction with Dr. Sutton, on cases of acute rheumatic fever treated with mint-water.

Another therapeutical paper was on the excellent results obtained by treating tapeworm with oil of male fern (1855).

In an early volume (1856) he published a remarkable paper on paraplegia, which by all competent judges is regarded as one of surprising sagacity and pathological insight. It ranks with the best contributions to our 'Reports' made by Fagge, by Wilks, or by Bright himself. Tumours of the cord, spinal meningitis, tabes dorsalis, are each dealt with in a masterly fashion, and with the pregnant brevity which appears now to be almost a lost art. This was followed by other papers continuing the subject in the 4th and 7th volumes of the third series. His publication on this subject led to a controversy with Dr. Brown-Séquard, in which Gull maintained the non-existence of what is strictly termed reflex paraplegia, arguing that when paralytic symptoms follow disorders of the bladder or similar peripheral lesions they are always accompanied by histological changes in the cord.

His Gulstonian Lectures, which unfortunately were never

separately published, interested him in the subject of cerebral abscess, upon which he published an admirable article in these 'Reports' (third series, vol. iii), and (again in conjunction with Dr. Sutton) the chapter dealing with the subjects in Reynolds' 'System of Medicine.' As a piece of purely pathological work this is as good as it can be.

Also with Dr. Sutton's help, he published the well-known paper in the 'Medico-Chirurgical Transactions,' on the condition of the arteries in chronic Bright's disease, which they called "Arterio-Capillary Fibrosis." The subject is still a difficult one, and much remains to be done before the old problem of the relation between the disease of the kidney and the concomitant vascular changes which puzzled Bright himself can be regarded as settled. But Gull and Sutton made, at all events, this step in advance: they showed that chronic Bright's disease affects the peripheral vascular system generally, and is far from being a mere local affection of the kidney.

Other papers, also in our 'Reports,' were upon such varied subjects as "Occlusion of the Carotid and Innominate Arteries," "Obstruction of the Abdominal Aorta," "Aneurysm of the Cerebral Arteries" and "Thrombosis of the Superior Mesenteric Vein," on "Pityriasis Versicolor," and on "Factitious Urticaria," on "Fatty Stools in Mesenteric Disease," and on "Pneumonia from Implication of the Pneumogastric Nerve." The "Report on Cholera" which, in conjunction with the late Dr. Bailey, he drew up for the College of Physicians in 1854, exhibits his industry and judgment rather than his more brilliant qualities.

A paper read before the Clinical Society on "Anorexia Nervosa" is a valuable contribution to practical medicine.

Sir William Gull's last important work was "On a Cretinoid Condition in Adult Women," a paper also published in the Clinical Society's 'Transactions.' It contains a brief but complete account of a hitherto undescribed condition, which has since attracted great interest under the more distinctive but less accurate name of myxœdema. As in other cases of clinical observation, it is remarkable that a disease which no one had recognised before its existence and characters were established by Gull has since been found to

exist not only in this country and on the Continent, but in America and Australia. Moreover, he not only caught its characteristic *facies* and recognised its importance, but in the name under which he described it he indicated its true pathological relations, doubtful and obscure for some time, but by recent investigation established beyond reasonable doubt.

Long before Wunderlich had shown the characteristic curves of temperature in fever, Gull had learnt the pathological lesson now everywhere recognised, that fever is a process, with a beginning, an ingravescent stage, an acme, and a gradual subsidence. He used to enforce this doctrine in his lectures by drawing curves on the blackboard,—a long, gentle, and irregular one for typhoid fever; a shorter, more abrupt, and more regular curve for typhus and for scarlet fever.

On the whole it may be said that scarcely any living physician has made so many, so important, and so varied original observations. Several of the papers above mentioned were, to use an expressive German word, "bahnbrechend." The conciseness of the language, and the unobtrusive manner of introducing the new observations, should enhance rather than diminish the credit of the author.

One of Gull's sayings has come true in the experience of many of his pupils. He said to the clinical assistants, "However clever you are, there are three diseases that you are sure to overlook in the course of your practice. They are phthisis and syphilis and itch."

He would say, "Medicines do most good when there is a tendency to recovery without them."

I once saw a lady who was very ill, and he was asked to come also. When we met, her husband with many apologies gave us a telegram he had received from her country doctor advising an external and internal remedy which he thought should be tried. Sir William read the message, and then answered, "Tell him that we are giving the one, and that the other would be of no use."

He once met a practitioner on a case of rheumatism in which he detected a pericardial rub. He said nothing of this to the patient's friends, but approved the general treatment,

and they came away together. "Oh, Dr. Gull, it was very good of you not to let them see I had made that dreadful oversight. I cannot think how I can possibly have failed to detect the pericarditis." "Never mind," said Gull, "it is just as well, for if you had detected it perhaps you might have treated it."

He had been attending a poor patient with heart disease, and after his death was extremely anxious for a post-mortem examination. With great difficulty this was granted, but with the proviso that nothing was to be taken away, and the sister of the deceased patient, a strong-minded old maid, was present to watch the proceedings. Gull saw that it was hopeless to conceal anything from her, or to persuade her to leave the room. He therefore deliberately took out the heart, put it in his pocket, and looking steadily at her said, "I trust to your honour not to betray me." His knowledge of character was justified by the result, and the heart is now in Guy's Museum.

He had a true scientific hatred of vague generalities, unverified conclusions, and superficial explanations. "How," he would say, "can you understand the 'constitution' of a patient when you have never examined his urine?"

A hypochondriac, who had been to several doctors of eminence and was dissatisfied with them all, came to 74, Brook Street, and after a brief examination begged to know the result. "Sir," said Dr. Gull, "you are a healthy man who is out of health." "Yes," said the patient, "that's exactly it; but why didn't the other doctors find it out?"

A medical student, afterwards a well-known physician, once consulted him for severe recurrent hæmaturia. Gull heard the history, examined the urine and the patient, and at last said, "You have no disease of the kidneys and you need not be anxious. Many young people are subject to bleeding of the nose. You have renal epistaxis."

A favourite remark of his was, "Savages explain, science investigates," which he enforced by remarking that every barbarous nation had a complete explanation of the cause of eclipses and other striking natural phenomena.

Sir William Gull always upheld a high standard of aim in the profession. He tried to impress his own personal feeling

of profound self-respect upon his pupils and upon the profession generally. Like his friend and colleague Sir William Jenner, he was always ready to uphold the true dignity of the profession, not in abstaining from the humblest services for the benefit of a patient, for no one was so observant and minute as he in these respects; nor in seeking after titles or scheming for precedence, for in Aristotle's language he did not much care for honours, inasmuch as he considered all honours beneath his deserts.[1]

He was fanciful in his ways of putting things, and sometimes perhaps affected singularity in this particular. A motto which he invented for himself pleased him so much that during the leisure of his last illness he not only had a seal engraved with the words, but had them printed on a slip of paper as a kind of memento. It was—

Conceptio Dei,
Negatio mei,
Ratio rei.

This was quoted by one of the newspapers after his death, but running the three phrases into a line their mutual dependence was lost. The true exposition was this: all knowledge begins with materialism, with scepticism, with getting at actual bare objective facts, the thing as it is—the investigation of phenomena, the linking together of necessary cause and effect, that is, the intellectual part of knowledge; and unless that is the foundation, morals and religion have no true and firm basis.

Next comes ethical knowledge, which consists essentially in self-denial, self-restraint, recognition of the overweening importance which we all give to the individual, and especially to one individual in comparison with the whole.

Lastly, as the crown of mental development, the fruit of

[1] "The magnanimous man is little moved by honours or by dishonours, for he regards the honour that is paid him as his due, and any contumely as undeserved. For which reason he is apt to be thought supercilious" ('Eth. Nich.,' iv, iii, 17, 18). To the same purpose Swift writes: "Vain men delight in telling what honours have been done them, what great company they have kept, and the like, by which they plainly confess that these honours were more than their due, and such as their friends would not believe if they had not been told; whereas a man truly proud thinks the greatest honours below his merit, and consequently scorns to boast."

knowledge, and the result of self-discipline, spiritual truth is discerned.

After he had spent his summer holiday at a place in Northumberland not far from a fishing village, he said to the writer, "I was very much interested in talking to the fishermen there. They are a fine set of men physically, and they are most of them brave, serious, and religious. Now I understand why fishermen were chosen to be apostles."

On the Senate of the University of London, in the General Medical Council, and at the College of Physicians Sir William Gull's voice was always in favour of science, of investigation, of advance and improvement. He was liberal in the old-fashioned sense of the word, disliking privilege and what he used to call "bumbledom." He took an active part in the defence of scientific medicine against the unscrupulous attacks of so-called humanitarians. He gave learned and well-considered testimony before the Royal Commission upon experiments on living animals. In union with Sir James Paget, Sir William Bowman, Dr. Wilks, Sir Joseph Lister, the late Dr. McDonell of Dublin, and the late Dr. Moxon, he broke through a wholesome rule which forbids professional men from writing in lay periodicals, and contributed a powerful article to the 'Nineteenth Century,' upon "Vivisection," of which the highest praise is that it was not unworthy of those by Bowman and by Paget, which it accompanied.

In conjunction with these two eminent surgeons, with Sir William Jenner, Sir Joseph Lister, Mr. Hutchinson, and the other leaders of the profession, he took an active part in the formation of the Association for the Advancement of Medicine by Research, which has done much to mitigate the mischief caused by legislation.

He gave important evidence before the Committee of the House of Lords upon the subject of intemperance, and gave a thoughtful and valuable address when he once presided at a meeting of the Charity Organisation Society.

He had a good knowledge of literature, and though not a scholar, read Latin and German authors with pleasure. George Herbert was a favourite author, particularly those pregnant lines of wisdom which form 'The Church Porch;' and the last words he read immediately before his fatal attack

were the fine imaginative lines of Vaughan, on "Departed Friends."

He took a deep interest in philosophical questions, and greatly admired his brilliant colleague, James Hinton, to whom, as to so many younger men, he afforded liberal help in practice.

Sir William Gull had enjoyed remarkable health throughout the whole of his life. Excepting an attack of enteric fever, he seems scarcely to have had any serious illness down to October, 1887. He was then at his house in Scotland, intending almost immediately to return to work after his autumn holiday, and while walking alone in the grounds was seized with paralysis. He did not lose consciousness, but fell on one knee and was able to walk to the house with assistance. He recovered slowly and returned to town after a few weeks. The significance of this attack of right hemiplegia with aphasia was obvious to the patient. He had shortly before had a warning of liability to hæmorrhage from degenerated vessels in a sudden and somewhat obscure attack of hæmoptysis without any sign of pulmonary or cardiac disease. His characteristic remark to the writer when he congratulated him upon his recovery was, "One arrow has missed its mark, but there are more in the quiver." He added, "I shall make no sudden change, and shall not give out that I will never see a patient again. The less one prophesies of oneself or other people the better, but I do not suppose that I shall do any more practice."

He took a house at Eastbourne, and the following season near Reigate, and made what is called a good recovery. But on several occasions he was again attacked with less severe though unmistakable repetitions of cerebral hæmorrhage. There was temporary loss of consciousness, sometimes slight eclampsia, and then temporary paralysis of one or the other side. Throughout, sensation was more affected than movement, the very condition on which he had discoursed in his Gulstonian Lectures. Moreover, as he said himself, he was subject to unaccountable and causeless but no less distressing apprehension, and vague feelings of uneasiness or distress. He preserved his mental faculties to the last, and spoke with

softened and resigned interest of the welfare of Guy's Hospital, "to which," he emphatically added, "I owe everything;" of public professional interests, and of his living and departed colleagues, friends and rivals. He referred to the inevitable result of his malady with admirable fortitude and submission. But his memory was beginning to fail, and though there was no increase of definite paralysis, power of locomotion gradually diminished, and capacity for reading and prolonged conversation was slowly lessened. It was therefore with feelings of thankfulness that those who best knew how sad a period of mental and physical decrepitude would prove, heard on the 28th January, 1890, that a final and painless attack of cerebral hæmorrhage, leading first to aphasia and shortly afterwards to coma, had ended the last scene of his useful and laborious life.

He was buried on the 3rd of February, a warm and beautiful day, with the sunshine and blue sky giving promise of the advent of spring, and harmonising with the hope of which the yearly return to life of things inanimate is the promise and the type. He was laid beside his father and mother in the churchyard of his native village, and was followed to his grave by the President of the College of Physicians, the Vice-President of the College of Surgeons, Sir James Paget, Sir Henry Acland, and a crowd of former colleagues and pupils, old friends, patients, and servants.

So well and in such esteem was ended a long and prosperous life, full of lessons which were taught both by precept and by example.

REPORT OF CASES

OF

ANTHRAX OR MALIGNANT PUSTULE

UNDER THE CARE OF MR. DAVIES-COLLEY;

WITH NOTES UPON THE TREATMENT OF THIS AFFECTION BY THE EXTERNAL AND INTERNAL USE OF IPECACUANHA.

By N. DAVIES-COLLEY.

In 1882 I published in the 'Med.-Chir. Trans.'[1] an account of two cases of external anthrax which I had recently treated in Guy's Hospital, together with a table of all the cases of this disease which Mr. Symonds, who was then Surgical Registrar, and I, had been able to find in our records from the year 1873, when, so far as I know, the first example was observed, to the date of the publication of the paper.

As the disease appears to be rare in this country, and as, when it does occur, it is apt to be confounded with less dangerous affections, I think that a short record of the cases which have come under my care since the publication of my paper in the 'Med.-Chir. Trans.' may be useful. It would appear that the disease is decidedly increasing in the south-eastern district of London, for whereas we were unable to find more than seventeen cases in the whole of our hospital records between 1873 and 1882, the number

[1] Vol. lxv, p. 237.

admitted into my wards alone, presumably, therefore, only one fourth of those actually treated, during the last seven and a quarter years have been thirteen.

My chief reason, however, for publishing these cases at the present time is that for the last two years I have been trying a new plan of treatment, viz. the internal and external use of ipecacuanha powder, and that I wish to call attention to the success which has hitherto followed the employment of this remedy.

The reports are chiefly taken from notes made by myself at the time, but I am also indebted for many particulars to the gentlemen whose names I have prefixed to the cases.

Case 1. *Anthrax of cheek; constitutional infection; excision; recovery.*[1] (Reporter, Mr. A. E. Taylor.)—Albert B—, æt. 43, employed in a leather warehouse in Tooley Street, was admitted under my care on February 18th, 1883. On February 15th he had scratched his face with a hide carried by a fellow-worker. That evening he noticed a swelling about the size of a split pea on his chin, and he had a rigor. He was unable to sleep that night. The swelling gradually increased. He could take no solid food, and in the night he was delirious. On the 18th he began to cough. There was no diarrhœa.

On admission he was a strongly framed, well-nourished man, but extremely weak, and unable to stand. Just behind the right angle of the mouth was a swelling rising abruptly from the adjacent skin to the height of about $\frac{1}{6}$ of an inch. Its surface was nearly flat, but the central part was depressed, and about $\frac{1}{20}$ inch below the surrounding level. It was of a bright orange-crimson colour, smooth, shining, and somewhat pellucid, while the concave central depression was covered by a thin purplish-black scab. Its dimensions were from before backwards $1\frac{1}{8}$ inches, from above downwards $\frac{13}{16}$ inch. The skin around was somewhat swollen, but little if at all reddened. There was considerable swelling of the soft parts from the corner of the mouth to $1\frac{1}{2}$ inches behind the mastoid process, and from the lobule of the ear to below the level of the angle

[1] This case has already been published in the Pathological Society's 'Transactions,' vol. xxxiv, pp. 291, 1883.

of the jaw. Two or three large glands could be felt indistinctly below the parotid gland; the largest was about one inch long. The eyelids were not affected. The swelling was not painful; he likened the sensation in it to the "gnawing of a mouse." Under chloroform the tumour was excised and the exposed surface cauterised. Numerous bacilli were found in the bloody serum which oozed from the punctured surface of the swelling. The wound was dressed with boracic acid ointment, and quinine in 5-grain doses was given three times a day. He made a good recovery, and went out with the wound nearly well on March 20th. Two days after the operation he was troubled with bronchitis. The viscid sputum was examined, and in it were found large bacilli, like those from the vesicles, and the next day a few similar bacilli were found in his urine. Nearly three weeks after his admission he had some diarrhœa, and passed blood and mucus with the loose motions. Similar bacilli were found in the fæces. It is probable, therefore, that he had some secondary anthrax foci in his intestinal tract, such as are frequently seen in patients who die from this disease.

Case 2. *Anthrax of right cheek; excision; extension of disease; application of Potassa Fusa; recovery.* (Reporter, Mr. F. Lever.) —Walter E. N—, æt. 28, a labourer at Fenning's Wharf, was admitted into Guy's Hospital on December 24th, 1883, with anthrax of the right cheek. Five days ago he had to handle wool from South Australia. Two days later, on the 22nd, he noticed a pimple on his right cheek, which itched a good deal. This has grown, but he has not suffered much pain from it. Eats and sleeps well. For six months has been a total abstainer. He is well nourished and apparently in good health, but rather pale. Two inches below the right tragus and half an inch in front of it is an indurated eschar, rather more than $\frac{1}{4}$ inch in diameter, and raised about $\frac{1}{10}$ inch. Its centre is purplish black, dry, and slightly depressed, and measures $\frac{1}{8}$ inch across. Around is a single row of vesicles, and beyond a red induration with a sharply defined external margin. The diameter of this red induration is from $\frac{1}{2}$ to $\frac{5}{8}$ inch. There is some swelling and a slight flush, with no definite margin, over an area of skin 2 or 3 inches across, ex-

tending from the tragus above to the level of the chin below, and from the back of the ear nearly to the corner of the mouth. No enlarged glands can be felt.

A disc of skin ¾ inch in diameter was cut away without an anæsthetic. The operation gave him very little pain. Iodoform was dusted on, and boracic lint applied. He was given Mist. Hydrarg. Perchl. ℥j ter die. Characteristic bacilli were found in abundance in the scrapings of a section of the eschar.

Two days later great swelling had developed on the right side of his face. The right eye was closed, the corner of the mouth distorted, and the lips greatly swollen. There was frontal headache and retching, but no vomiting. His temperature was 102·6°, his pulse 100. The wound looked dry and shallow. He was taking quinine in 10-grain doses three times a day, and also 4 oz. of brandy. I rubbed the wound very thoroughly with Potassa Fusa, destroying its base to a depth of about ⅙ inch, but very little of the margin. The operation was rather painful. In the afternoon he vomited twice, and his temperature rose to 104°. The next day he had had no sleep, but felt somewhat better, and his temperature was lower. He still vomited occasionally. The swelling had somewhat increased. It extended from the top of the temporal ridge to the level of the cricoid cartilage, and from the back of the ear to the anterior middle line. The right eye was closed, and the right half of the lips much swollen. The colour of the general swelling was a somewhat livid pink with no defined border. It was moderately firm but not hard. There were no vesicles upon it. The elevation of the swelling was about half an inch in the centre, and it sank gradually at the sides to the normal level. There was a good deal of aching pain in the occipital region, but not much in the swelling. On the fourth day he was better. The nausea had gone, and he had slept well after a morphia injection. The face was less swollen. The bowels were loose. No bacilli could be found in any of the excreta. From this time he steadily improved, and he was discharged with a small healing wound on January 29th.

Case 3. *Charbon of neck; excision and application of zinc*

chloride; death. (Reporter, Mr. L. Bidwell.)—Isaac R—, æt. 48, a warehouseman, was admitted into Guy's Hospital on 16th February, 1884, with malignant pustule on the upper part of the right side of his neck. He was employed in some leather works, and had recently been handling raw hides from Bombay. Three days ago one of his mates said to him, "What is the matter with your neck? it looks red." Next day there was a distinct swelling there. He worked till 6 p.m. the day before admission. Last night he slept well, but he awoke with a difficulty of breathing. About 11 a.m. he vomited two or three times, bringing up chiefly phlegm. He has a poor appetite. He was brought up to the hospital in a cab, but thinks he could have walked a mile or two.

On admission he was a stout man, with a florid but somewhat livid complexion. About one and a half inches below the right ear was an elevated induration about 1 inch in diameter. The centre of this was purple. Around was a border of large vesicles about $\frac{1}{6}$ inch wide, and this was surrounded by a red margin, which was but little raised above the softer tissues around, as they were themselves considerably swollen. The sides of the face and neck were much swelled, the swelling extending from above the ear well into the submaxillary region of the other side. The œdema reached backwards and downwards to the upper part of the scapula, and in front to below the nipples. Over the cheek the greatest elevation was nearly an inch above the ordinary level, but the eyelids were unaffected. There was some redness of the surface, but this may have been due to the linseed meal poultice which he had applied.

After injecting $\frac{1}{4}$ gr. of morphia I cut out a circular patch of skin, $1\frac{1}{4}$ inches in diameter, including the anthrax eschar, and filled the wound with a mixture of chloride of zinc and flour, 1 in 4; boracic lint dressing was then applied. He did not feel much pain. Quinine in 5-grain doses at intervals of four hours was ordered, and brandy mixture, ℥j every two hours. Numerous bacilli were found in the serum from the vesicles. During the night he vomited part of the quinine and of the brandy mixture. The next day there was a good deal of dyspnœa, and he brought up much phlegm. His hands were bluish, his pulse rapid and almost imperceptible. He lay in

a half-sleeping condition. Brandy enemata were given, but he gradually sank, and died at 5.45 p.m.

Upon post-mortem examination the next day he was found to have extensive œdema of the mediastinum. The lungs were collapsed, dry, and leathery from want of air. No fluid was found in the pleural cavities. The stomach was "excessively mammillated, and had in addition a large number of peculiar slashed ulcers in all parts and of all sizes. They looked just as if someone with a sharp knife had slashed here and there. Some of them were healed; others, however, small ones, at the pylorus, were distinctly ulcers, and one had a considerable amount of vascularity about it, and its edge was yellow. It was a question whether there might not have been some gastric anthrax developing. If so, it was extremely early." Nothing abnormal was found about the intestine. The spleen was peculiarly bloodless, dry, pale, and flaccid.

Case 4. *Anthrax of left cheek; excision and application of zinc chloride; recovery.* (Reporter, Mr. G. T. Rawnsley.)—George R—, æt. 33, admitted on March 17th, 1884, with charbon of left cheek. He works with the skin-brokers, Messrs. Anning and Cobb, Tooley Street.

On 11th he noticed two pimples on the side of his face, one at the corner of the mouth, the other where he now has the swelling. He had been handling hides from Bombay and the Cape. He applied some sticking-plaster, and one pimple disappeared. For two days the other has been swelling rapidly. He has not been ill till this morning, when he felt faint. About halfway from the lobule of the left auricle to the tip of his chin is a flat indurated elevation rather more than $\frac{1}{2}$ inch in diameter; it is red and raised $\frac{1}{10}$ inch above the adjacent skin. The greater part consists of a ring of vesicles round a slight central depression of a purplish-brown colour; around this is a puffy swelling with a slight flushing of the skin from the corner of the mouth to his mastoid process, and from the zygoma to the clavicle. There is no pain, but only a sense of tingling and weight about the eschar. There is also a slight tenderness about a swollen gland $\frac{3}{4}$ inch in its longest diameter, which may be felt near the posterior border of the mastoid process. He felt faint this morning,

and has had some pain in his stomach. Otherwise he feels well. Pulse 112, temp. 100°, resp. 22.

Under ether I cut out the eschar with about $\frac{1}{6}$ inch of the adjacent integument. Some small vessels had to be ligatured. The wound was then filled with a mixture of zinc chloride and flour (1 in 4), and boracic acid lint was applied. Abundant bacilli of anthrax were found in the bloody serum from the wound. He was ordered Pulv. Quiniæ gr. x ter die, and 4 oz. of brandy daily. Low diet with two eggs. He made a rapid recovery; the swelling of the gland went down without suppuration, and he went out nearly well on March 26th.

Case 5. *Anthrax of neck; excision and application of chloride of zinc; recovery.* (Reporter, Mr. S. Wacher.)—Michael W—, æt. 26, a labourer at a skin warehouse (Messrs. Flack, Chandler, and Co., Stoney Lane), where he is frequently brought into contact with foreign hides, most of them imported from China, was admitted on March 18th, 1884, with charbon of the right side of the neck behind and below the mastoid process. He was a big, muscular fellow, and he had been drinking rather heavily of late. On the 15th he noticed a pimple upon his neck, and some aching. On the 17th it began to swell. He has had no constitutional disturbance.

In the upper third of the right posterior triangle there was an elevated patch, consisting of a zone of low whitish vesicles around a slightly depressed brownish-purple centre, and an outer zone of red induration. The whole patch was $\frac{5}{8}$ inch in diameter, and it was elevated about $\frac{1}{6}$ inch above the adjacent skin. There was considerable swelling and œdema around, but no enlarged glands could be felt. I excised the eschar under ether, and applied zinc chloride and flour with boracic acid lint, and quinine sulphate was given in 5-grain doses three times a day. He recovered rapidly, and went out on March 30th.

Case 6. *Anthrax of hand; excision; œdema of back of forearm; incision; recovery.* (Reporter, Mr. S. Wacher.)—Walter S—, æt. 37, a tanner from Messrs. Beresford and Co., St. Olave's Wharf, was admitted April 9th, 1884, with charbon of the right hand. He had been handling China and Bombay hides lately. Two days ago he noticed a pimple on the back of his hand, which he scratched. It grew, and the parts

around swelled rapidly, but he has had no pain in the part. He has never heard of any similar affection among the workmen in the warehouse for which he works.

Just above the metacarpo-phalangeal joint of his right middle finger is an elevation of about $\frac{1}{16}$ inch with a diameter varying from $\frac{1}{4}$ inch to $\frac{3}{8}$ inch. It was red and indurated at the sides, blackish in the middle. The centre looked as if there had been a vesicle there which had burst. There were no vesicles at the sides. The back of the hand was very œdematous, and the swelling extended an inch up the forearm, and also for a short distance upon the dorsum of the fingers. The supra-condyloid gland was enlarged. There were no constitutional symptoms.

Under ether the eschar was cut out by a circular incision three quarters of an inch in diameter. It was dressed with iodoform powder and carbolic gauze. In the scrapings from the surface of the eschar characteristic bacilli were plentiful. The next day the forearm was swollen nearly as high up as the elbow, and there was some flushing of the skin. He had not been able to sleep all night, and he felt faint in the morning. In the same evening he became much worse. The swelling of the arm had increased much, and there was great pain in the hand and arm. His temperature had risen to 104·2°, and his pulse was 120. An anæsthetic was again given, and Mr. Symonds made numerous incisions in the forearm and hand. A large quantity of serum, mingled with blood, flowed from them. A few characteristic bacilli were found in the fluid. Some carbolic acid lotion (♏xlv) was injected subcutaneously in the arm, and the operation wound was rubbed with Potassa Fusa. Salicylate of soda was given in 20-grain doses every two hours. The patient was much relieved, and the feverish symptoms at once disappeared. Rapid recovery took place, and the patient went out nearly well on March 26th.

Case 7. *Anthrax of right side of neck; excision; recovery.* (Reporter, Mr. F. F. Foster.)—John McS—, æt. 34, a warehouseman from Messrs. Culverwell, Brook, and Cotton, was admitted into Guy's Hospital December 22nd, 1884. He had been working with Calcutta goatskins for some time

previously. On the 19th he noticed what he called "a blind boil" on his neck. The swelling had since greatly increased. He had had a cold with running at the nose for a week. His appetite had been bad on the 19th and 20th, but he had slept well.

On the right side of his neck just below the ear was an elevation $\frac{1}{3}$ inch in diameter and $\frac{1}{10}$ inch high. In the centre it was depressed, and it looked as if the secretion had dried with dirt or flue from the coat upon it. It was somewhat pellucid at the margin, but there were no distinct vesicles. Around was a diffused redness with a swelling, especially for two inches downwards and forwards from it. It was not very tender. There was some swelling of the cervical glands.

A disc of skin $\frac{5}{8}$ inch in diameter was removed without an anæsthetic. Zinc chloride and flour were applied, and a dressing of boracic acid lint. He was ordered Mist. Quiniæ ℥j ter die. The scrapings from the eschar showed numerous large bacilli. He made a good recovery, and went out nearly well on January 16th, 1885.

Case 8. *Anthrax of neck; excision; application of zinc chloride; extension of disease; dyspnœa; laryngotomy; death.* (Reporter, Mr. C. J. Prime.)—George R—, æt. 45, potato warehouseman next door to a hide warehouse at Topping's Wharf, was admitted under my care into Guy's Hospital on March 10th, 1887. He noticed on the 6th a painful swelling on the left side of his neck, which has grown steadily since. He says that they have lately pitched a great number of potatoes from the warehouse among the hides.

About two inches below the left ear is a characteristic anthrax swelling $\frac{1}{4}$ inch in diameter. There is also hard œdema around from the middle line in front nearly to the nape of his neck, and from his ear nearly to his clavicle, the elevation of the swelling above the normal level being about $\frac{1}{4}$ inch. He looks fairly well but a trifle pale, and he says that he slept very little last night. Twenty drops of 5 per cent. solution of cocaine were injected in five places, and the eschar was excised. Chloride of zinc and flour were applied, and a mixture containing eighty drops of Liq. Hydr. Perchl. in each dose was ordered to be taken three times a day. Next day

there was much swelling over the side of his face, reaching down to the nipples and across the middle line of his neck, and there was also some pain around the wound. He had difficulty of breathing last night. When I saw him he was breathing quietly without any stridor, and there was no lividity. Pulse 130. He sat up in bed well. The swelling was faintly flushed. Three longitudinal incisions were made with a knife a quarter of an inch deep in the vicinity of the wound. He was ordered Sodii Sulphitis gr. x ex aquâ every three hours, and I injected subcutaneously twenty minims of carbolic lotion between 10 and 11 a.m. into the parts around. Numerous large bacilli were found in the serum from the surrounding œdema. None could be seen in the thick mucus which he spat up.

The second day the pulse was 150 and hardly to be felt. He was quite sensible. He had been vomiting, but there was no pain in the stomach. There was more lividity of the face, and the swelling had increased so much as to close his left eye.

On the 13th the pulse was about the same, and difficult to feel. Respiration about 29. He was sensible. The redness and œdema of neck were unchanged. The wound made by the removal of the eschar was dry and depressed, and surrounded by thin-walled irregular clear blebs, more like those of erysipelas than of charbon. I rubbed Potassa Fusa into the wound and cauterised the skin superficially for a quarter of an inch all round. Numerous bacilli were found in the serum from the blebs. The sulphite of sodium had been stopped, as he vomited after each dose.

On the 14th he complained of difficulty of breathing. There was still great swelling of the neck. The right eye was not affected, but the left was tightly closed. Pulse was rapid, 120, but hardly perceptible. Respiration 28. His chest was fairly resonant, but very little respiratory murmur could be heard at the back of the right side. He was quite sensible, and could sit up. About 5.30 p.m. his breathing became very much embarrassed, and with some difficulty laryngotomy was performed by the house surgeon, Mr. Burghard. For a time the relief was great, but he gradually sank afterwards, and died early on the morning of the 15th.

At the post-mortem examination made next day he was

found to have œdema glottidis. The whole of the surface of the brain was thinly covered by meningeal hæmorrhage. The lungs were dark, with black patches of hæmorrhagic congestion. A few subserous ecchymoses were found, and some passive effusion in the pleura. There were also some distinct deposits in the upper part of the small intestine, each surrounded by an area of congestion, and with their centres sloughing and ulcerated.

A year later I saw in the 'Lancet'[1] an interesting paper by Dr. Edwin B. Muskett, which induced me to employ ipecacuanha in the treatment of these cases.

Dr. Muskett, who is in practice in South Africa, states that splenic fever is common among the cattle there, and that human beings are not unfrequently infected by them. His plan of treatment is to use ipecacuanha, applying it externally with water in a mixture of the consistence of cream, and administering it internally at the same time. During fifteen years he has treated fifty cases in this manner without a single death. When I saw this account it seemed to me that one great advantage of this mode of treatment was that by administering such a remedy as powdered ipecacuanha internally it was probable that the drug in this form would not be immediately absorbed, but that the greater part of it would pass for some distance down the alimentary canal. If, therefore, it has any specific action upon the bacilli, it would be brought into contact with the small black sloughs and the ulcers which are so frequently found in fatal cases upon the mucous membrane of the stomach and intestines, and which are probably the cause of the vomiting, diarrhœa, and stomach pain of which my patients have so frequently complained. This probability is further increased by the consideration of the great benefit which results from the use of large doses of this drug in chronic dysentery, for it seems to me most likely that this was attributable to some specific action which it exerts in its passage down the intestinal tract upon the ulcers in which it may be presumed that the poison of that disease is chiefly located.

Dr. Muskett does not resort to excision of the cutaneous

[1] Vol. i, p. 269, 1888.

eschar. I have not as yet ventured upon omitting this precaution, which has been of so much advantage in the great majority of the cases hitherto recorded at Guy's Hospital. I intend, however, in future cases to proceed cautiously in the direction of removing less and less of the eschar and the adjacent skin. It will be seen, however, that in the four following cases in which I have hitherto had the opportunity of using the remedy I have only ventured upon applying the ipecacuanha after excision, in place of the treatment with zinc chloride or the other antiseptics which I have usually employed.

Case 9. *Anthrax of right temple; excision and treatment with ipecacuanha; recovery.* (Reporter, Mr. H. B. Bolus.)—Charles P—, æt. 22, a salter in the hide trade, at the works of Messrs. Culverwell, Brook, and Cotton, was admitted under my care into Guy's Hospital on June 27th, 1888, with anthrax behind the right eye. On the 21st, soon after they had received a new consignment of hides from Bombay, he noticed a pimple behind his right eye, which he tried to get rid of by taking two ounces of Epsom salts. The swelling had grown steadily, but without pain; there had been no impairment of appetite or sleep.

About one inch behind the outer corner of the right eye was a somewhat pellucid reddish elevation $\frac{7}{16}$ inch across, with a depressed black dry centre and a red œdematous base around. The eyelids were somewhat œdematous, but could be separated. A disc of skin $\frac{5}{8}$ inch in diameter was removed after cocaine had been applied for five minutes. Characteristic bacilli were found in the scrapings. The wound was dressed with ipecacuanha powder and boracic acid lint, and 5-grain doses of the same powder were given to him every four hours.

On July 2nd the swelling of his eyelids had gone down, but the glands, viz. one below the zygoma and in front of the auditory meatus, and the other behind the angle of the jaw, were still enlarged as before. He had gone on taking the ipecacuanha powder regularly, and after the second day it caused no sickness. He made a rapid recovery, and went out on July 5th, 1888.

Case 10. *Anthrax of front of neck; excision; ipecacuanha treatment; some sloughing of soft parts; recovery.* (Reporter, Mr. A. Leigh Allworth.)—William J—, æt. 21, who works as a labourer at Anning and Cobb's, Morgan Lane, Tooley Street, was admitted under my care into Guy's Hospital on March 11th, 1889, suffering from anthrax of the front of his neck. He has lately been working in Bombay hides and kangaroo skins. Yesterday morning he noticed a swelling. His collar felt tight, and there was some irritation in the neck. Later in the day he felt unwell, and he went to bed at eight o'clock. He slept well, but has not been able to eat anything to-day.

On admission his temperature was 102°, and his pulse was quick and irregular; no vomiting. Characteristic eschar about ½ inch in diameter, and elevated $\frac{1}{20}$ inch in the middle line of the front of the neck, about halfway between the chin and the sternum. The front of the neck and the parts around were much swollen, giving him the appearance of a man with a great double chin. The swelling extended as low down as the third costal cartilages. The skin was slightly flushed. Under ether, during the administration of which he became very blue, a piece of integument 1¼ inches by ¾ inch was removed. The wound was dressed with a quantity of dry powdered ipecacuanha, covered by a pad of boracic acid lint. Five grains of the same drug were given every four hours, and 10-grain doses of quinine were ordered if the temperature should rise above 102°. Brandy ℥iv daily. By the evening his temperature had risen to 104°.

The next day he was very feverish; his pulse 130 to 140, and his temperature had risen still higher. His head was clear, and he said he felt well, but complained of some difficulty in breathing. Ten-grain doses of quinine had been given him as well as the ipecacuanha. He had vomited after the quinine, but not after the ipecacuanha. The swelling had extended, and there was redness of the skin, but no lividity. No more quinine was given, but he went on with the ipecacuanha treatment. The next day he was considerably better, and the swelling was going down. He was sick once in the morning, but not afterwards. About a fortnight later some phlegmonous inflammation occurred around the wound, and in the deep connective tissue at the right side of

it. It was dressed with compresses of boracic lint and hot boracic lotion, and some sloughing tissue came away. The wound soon granulated up, and he went out convalescent on April 3rd.

He states that he had an anthrax removed from his left thumb four years ago in the surgery of Guy's Hospital.

Case 11. *Charbon of neck; excision; ipecacuanha treatment; recovery.* (Reporter, Mr. F. Wallace Wilson.)—John B—, æt. 26, dock labourer, who has nothing to do with hides, was admitted under my care on December 12th, 1889, with anthrax under his chin.

He had been drinking heavily lately, and cannot give a clear history of himself. He thinks that he first noticed a pimple two days ago. It caused no pain nor itching, but grew rapidly. He slept well last night; to-day he has taken nothing solid, but only some stimulants.

On admission he was stout and strong-looking, but somewhat bloated in appearance. In the left submaxillary region, a short distance from the lower border of the inferior maxilla, is a well-defined, firm, tender swelling, $\frac{1}{2}$ inch in diameter and raised about $\frac{1}{10}$ inch. The centre is dry, brownish, and somewhat depressed, at the margin somewhat pellucid, but without any definite appearance of vesicles. There is a general swelling from the chin to the hyoid bone, and from an inch to the right side of the anterior middle line of the neck nearly to the lobule of the left ear. There is a faint flush on the surface of the skin with no defined margin. Under ether a disc of skin an inch in diameter was dissected out. Ipecacuanha powder was applied to the wound, with a covering of sal alembroth gauze, and 10-grain doses of ipecacuanha were given every three hours. Diet of milk, eggs, and beef-tea ordered.

December 13th.—Morning temperature 103°. The wound was dressed again in the same way. There was no diminution of the swelling. He felt comfortable, but complained of great thirst and some pain in swallowing.

14th.—His temperature rose to 104·2°. Quinine Sulph. gr. v given. He had seven loose motions to-day, and a little vomiting. Brandy ʒij daily, and Opii gr. j horâ somni.

15th.—Vomiting and diarrhœa less. Temp. 102°. He keeps down a good deal of milk. The œdema of his neck has spread upon the other side. Brandy increased to six ounces daily.

16th.—Fever less. Still more diarrhœa and vomiting, and no marked diminution of œdema. Pulv. Ipecac., gr. x, sextis horis.

17th.—Dose of ipecacuanha diminished to gr. v ter die, and to this morphiæ gr. ¼ added. Brandy ʒiv.

18th.—Swelling going down. Temp. 100°, pulse 114. Patient has not vomited for twenty-six hours.

20th.—Decidedly better. Temperature normal. Ipecacuanha powders left off.

21st.—Allowed to get up.

24th.—Wound still rather sloughy.

January 3rd, 1889.—Granulating wound ⅞ inch across.

11th.—Went out convalescent.

Case 12. *Anthrax of face; excision and ipecacuanha treatment; recovery.* (Reporter, Mr. F. Wallace Wilson.)—Charles P—, æt. 16, a healthy-looking labourer on the South-Eastern Wharf, was admitted into Guy's Hospital on December 15th, 1889, with a well-marked anthrax over the left cheek-bone. He had been carrying hides in vans since the 12th. On the 11th he noticed a swelling like a little boil, and he scratched it and knocked the head off on the morning of the 12th. He had also been handling hides three weeks before. His health continued good until the night of the 13th. Since then he has slept badly, but has taken his food well and has not been sick.

On admission.—Over the left malar bone is a nearly circular swelling, flat and about ⅞ inch in diameter, depressed in the centre, with a zone of well-marked vesicles near the border. There is soft œdema with very little redness over an area 7 or 8 inches in diameter, extending from 1½ inches above the zygoma to the level of the hyoid bone, and from the front of the cheek to the back of the mastoid process. The elevation of the general swelling is about half an inch at the highest point. There is some enlargement of the upper cervical lymphatic glands. Pulse 116, temp. 100°. Feels out of sorts. Some pain in the neck, but not elsewhere.

A piece of skin was excised under ether about $1\frac{3}{8}$ inches in diameter, and ipecacuanha powder was given in the same doses and applied in the same manner as in the previous case. To take milk, beef-tea, and four eggs daily.

On the 16th he was very sick. Temperature over 100°, but he was not suffering much pain.

On the 17th still very sick. Doses of ipecacuanha were diminished to 5 grs., with the addition of $\frac{1}{6}$ gr. of morphia three times a day. The wound was dressed for the first time. There was still a good deal of swelling behind the jaw, and a gland could be felt about one inch in the longest diameter.

18th.—Temperature below 100°. Has not been sick again. Pulse 80.

On the 20th the powders were stopped. He had taken them regularly up to this time.

On January 5th, 1890, he went out convalescent. The wound was healthy, and the last notice of the enlarged gland states that it was diminishing in size.

Case 13. *Charbon of neck; excision; internal administration of ipecacuanha; recovery.* (Reporter, Mr. J. R. Evans.)—James M—, æt. 24, a labourer at Dyster and Nalder's leather warehouse, was admitted on April 6th, 1890, with anthrax on the front of his neck. Mr. Symonds took care of the case in my absence, and has kindly permitted me to publish it in this series. A small pimple appeared on April 3rd under his chin, and his attention was called to it by a little irritation caused by his muffler rubbing against it. There was no pain in the swelling, which steadily increased in size until he came in.

On the 6th, when he was admitted, there was a red induration nearly an inch across, with a ring of vesicles upon it surrounding a black slough in the middle line of the neck, a short distance below the chin. Some redness around, but no œdema. No enlarged glands felt.

Under an anæsthetic the eschar, together with a quarter of an inch of the adjacent skin, was excised. The wound was washed with carbolic acid lotion (1 in 20). Iodoform was dusted on, and carbolic and sal alembroth gauze applied.

Five-grain doses of ipecacuanha were given every four hours. This was left off on the 9th, and some quinine and iron were ordered. He recovered rapidly, and went out convalescent on April 19th.

The patient states that three men besides himself were attacked with the complaint.

Remarks.—I do not propose to discuss in this paper the etiology and diagnosis of anthrax, as I can add very little to what I have already written in my paper in the 'Med.-Chir. Trans.,'[1] and also in an article upon the disease in 'Heath's Dictionary of Surgery.'[2] My chief object is to call attention to the mode of treatment recommended by Dr. E. B. Muskett.

In all my cases there was no difficulty in recognising the disease. There was in each the well-defined area of induration, with its purplish or blackened dry central depression and its encircling ring of vesicles or pellucid infiltration. In most of them there was also considerable œdema of the integuments for some distance from the eschar. With one exception the patients had been in contact with hides, and the only one who had not was a wharf labourer, whose occupation would expose him to contact with various goods, and not improbably to that of bales of foreign hides. In Cases 1, 3, 4, 8, and 10 there was evidence of constitutional disturbance, but in some of them the symptoms were only slight, such as inability to sleep and loss of appetite.

In nearly all the cases the surrounding œdema had spread far beyond the primary eschar, so that the excision to which I resorted in each case must have left behind a considerable amount of infected tissue. In some there was also evidence of lymphatic absorption. Of the eight cases treated without ipecacuanha two died. In both of them the neck was the seat of the anthrax. I have before shown that the disease in this locality is especially dangerous. The reason for this would appear to be, firstly, the proximity of the larynx, which is apt to give rise to œdema glottidis (as seen in Case 8); and secondly, the disposition of the cervical fascia, which enables the inflammation to spread downwards into the mediastinum (as occurred in Case 3).

[1] Vide loc. supra cit. [2] Vol. ii, p. 2.

The last five cases were treated with ipecacuanha, Nos. 9, 10, 11, and 12 by the internal and external use of the powder, and No. 13 by the internal use for three days only.

All recovered, although three out of the five cases had anthrax of the neck. One of these cases, No. 10, was in great danger. He had distinct constitutional infection upon admission, and the next day after the operation the swelling had increased, and there was some difficulty of breathing. In him some local sloughing occurred in the connective tissue close to the wound. It would seem, therefore, that the ipecacuanha had in this case acted rather violently upon the neighbouring tissues. In the three other cases, however, in which it has been locally applied no such result has been observed. Possibly, therefore, this phlegmonous inflammation should be attributed rather to the virulence of the anthrax infection. In Case 6, for example, we have an instance of great inflammatory swelling running up the forearm after the removal of an anthrax from the back of the hand. This, however, was treated by free incision, and no sloughing followed.

A good deal of vomiting was noticed in some of the cases from the internal use of the ipecacuanha. We must not forget, however, that this symptom also occurred in other cases which were not treated in this way. It may be ascribed in some measure to the constitutional infection, one result of which is the development of secondary anthrax foci in the mucous membrane of the stomach and small intestine (vide Case 8). In Cases 11 and 12 the dose of ipecacuanha was probably too large, *e. g.* gr. x; hence the obstinate vomiting which followed its administration. But when the dose was diminished to gr. v, and guarded by the addition of morphia gr. $\frac{1}{6}$, as recommended by Dr. Muskett, it was taken without any inconvenience.

In order to ascertain the causes of the influence which ipecacuanha appears to exert upon anthrax, I asked my friend and colleague Dr. Washbourn to make some researches upon pure cultivations of the bacillus in his laboratory. Mr. Evans, his assistant, has kindly furnished me with a note, which I append to this paper, upon the result of this inquiry. It will be seen that, as far as regards the bacillus itself, the destructive effect of ipecacuanha is clearly proved. The spores of

the bacillus, on the other hand, are able to resist the influence of the drug, but as they are not found in the development of the disease in the human body this does not impair the efficacy of the ipecacuanha treatment.

Note on the Action of Ipecacuanha upon the Bacillus Anthracis, by Mr. Percy C. Evans.

Mr. Davies-Colley having found ipecacuanha a valuable remedy in the treatment of malignant pustule, I, at his suggestion, undertook a series of experiments to test its action on pure cultures of the Bacillus anthracis in the bacteriological laboratory at Guy's Hospital. The following is a summary of the method employed in these experiments, and of the chief results. Full details will be found in my paper in another part of the Reports.

The ipecacuanha root was procured from the dispensary in the form of a fine powder. Weighed quantities of this were added to test-tubes containing an abundant and pure growth of Bacillus anthracis in peptone broth. The contents of the tube were then gently mixed, and after the ipecacuanha had acted for a quarter or half an hour fresh tubes of culture media—broth, agar, or gelatin—were inoculated by means of a platinum loop. If the ipecacuanha had been added in sufficient quantity to kill the anthrax bacilli in the original tube no growth occurred in this second series of tubes; when they were not destroyed an abundant growth of the bacilli occurred in one or two days.

As a result of numerous experiments carried out on this method, it was found that ipecacuanha in quantities of 5, 4, 3, and 2 grains, added to tubes containing 5 c.c. of broth, invariably destroyed the vitality of all the anthrax bacilli present, and no growth occurred in the tubes inoculated, provided always that the bacilli contained no spores. With smaller quantities of the root varying results were obtained. Thus when one grain was added to a tube containing 5 c.c. broth, some of the tubes inoculated after a quarter to half an hour showed no growth, whilst in others an abundant growth

occurred. Quantities less than this, such as half a grain, had no action at all, even after acting for one hour.

It appears, therefore, that ipecacuanha has a very decided power of destroying the vitality of Bacillus anthracis when growing in broth and free from spores. On the other hand, the powder was found to have no effect whatever on the vitality of anthrax spores, even after acting for twenty-four hours. This is, however, of no importance as regards the therapeutic value of ipecacuanha, inasmuch as anthrax bacilli never produce spores in the tissues of a living animal.

Further experiments with the active principles of ipecacuanha have been made, and will be described in my other paper in these 'Reports.'

SARCOMA OF THE TONGUE.

By J. H. TARGETT, M.S.

In his monograph on 'Diseases of the Tongue,' which was published in 1885, Mr. Butlin remarks that "primary sarcoma of the tongue must be regarded as an exceedingly rare form of tumour." So little material was he able to collect, that he felt it quite useless to attempt to write an account of this disease; but he adds, "Now that attention has been drawn to its extreme rarity, it is possible that some instances which have been observed, and not yet recorded, may find their way into surgical literature." In this paper I propose to record a case which occurred last year in Guy's Hospital under the care of Mr. Howse, to whom I am indebted for permission to publish the notes, and to append abstracts of such cases as are reported in the various medical journals. An examination of the surgical reports of Guy's Hospital for the last twenty-five years has supplied but three cases, including that which forms the subject of this paper.

Case 1 ('Guy's Surgical Reports,' 1888).—Joseph P—, æt. 65, admitted under Mr. Howse for a swelling in the mouth, which he first noticed a year ago. It was then the size of a horse-bean, and was situated about the middle of the tongue; but it has gradually enlarged ever since, though without causing any pain. He says he has lost weight lately. Family history and previous history very good.

On admission there is in the substance of the tongue a

globular tumour, harder than the tissues of the tongue, and not fluctuating. It is most apparent on the left side of the frænum. This globular mass is separated by a groove from another swelling which extends along the left side of the floor of the mouth from the frænum to the last molar tooth, is softer than the former, and semi-fluctuating. These two swellings appear to be connected deeply in the submaxillary region, for with the forefinger on the floor of the mouth and the thumb under the ramus of the jaw they seem to move as one. In the submaxillary region, too, is an enlarged, freely movable lymphatic gland.

The patient can swallow easily and speak clearly, but the movements of the tongue are somewhat impeded by the tumour. There is no pain. The surface is not ulcerated.

Antisyphilitic treatment was adopted for three weeks, but as there was no visible improvement an operation was recommended. Under an anæsthetic the left cheek was incised from the angle of the mouth backwards for an inch and a half, the tongue was drawn forwards, and was divided with scissors along the median line for the anterior two thirds of its length, and the left portion removed, including the growth and the mass in the floor of the mouth. There was free hæmorrhage requiring many ligatures; afterwards the wound was painted over with iodoform in ether, and the incision of the cheek was closed.

The subsequent treatment consisted in painting over the raw surface in the mouth with iodoform every hour for the first day, and less frequently afterwards; and the patient was fed with milk through a tube passed over the back of the tongue. Within three weeks of the operation he was discharged quite well.

Fifteen months after the operation the patient was again examined, and a large mass found in the left submaxillary region, which had been slowly increasing in size. There was also a swelling below the zygoma on the right side of the face, which appeared to be a secondary deposit. In the floor of the mouth and the remainder of the tongue there was no recurrence. He complained of much discomfort in the chest, and had had a severe attack of bronchitis, but there were no physical signs of secondary disease in the lungs.

Examination of the tumour.—The parts removed by operation were found to contain two masses of new growth, one the size of a walnut occupying the site of the sublingual gland, and the other embedded in the substance of the tongue. A vertical longitudinal section of the tongue showed that the latter mass was spherical in shape, an inch and a half in diameter, and very soft in structure. It appeared moderately well defined, the muscular tissue being displaced by it; its upper limit was half an inch below the dorsal surface of the tongue; in front it nearly reached the frænum linguæ, while its outer surface was immediately beneath the mucous membrane of the floor of the mouth. There was no ulceration of the mucous membrane in any part. What has been described above as the sublingual portion was very closely connected with the mass in the tongue, and was probably an extension of the disease. Its relation to the sublingual mucous gland could not be determined by dissection or histological examination; hence it was suggested that the latter had either not been removed at the operation, or had already been destroyed by the growth.

Microscopic sections were prepared from different portions of the tumour, after the tissues had been embedded in celloidin. They were composed of small round cells, remarkably uniform in size, and held together by a little granular material. There was no true stroma, but the sections were permeated with thin-walled vessels which in some degree supported the cells. A few strands of muscular and fibrous tissue ran through the sections, and served to divide them up into large areas of cells, but were not sufficiently abundant to render the tumour firm. Within these strands one or two nerve-bundles were seen in transverse or oblique section. Further, among the cells themselves tiny pieces of muscular fibres were occasionally found, and the method of invasion by lines of cells between the muscle-fibres was easily made out. The sections were carefully searched for parasites, but without success. It was, therefore, concluded that the growth was a true round-celled sarcoma, which had begun probably in the connective tissue of the substance of the tongue, and gradually destroyed and replaced its muscular tissue; in other words, that it was a primary sarcoma of muscle affect-

ing the tongue, and resembling in its structure and development such new growths in other regions of the body.

Case 2 ('Guy's Surgical Reports,' 1867).—Albert W—, æt. 2, was admitted under Mr. Durham for a swelling on the tongue which his mother had first noticed seven weeks before. It had much increased in size of late, but there was no pain in it. The child snored loudly in its sleep. The family history was very good.

On admission.—There was a tumour on the dorsum of the tongue, to the left of the median line and about one inch and a half behind the tip. It was whitish, of a soft consistency, and measured one inch in diameter. There was no enlargement of the glands in the submaxillary region. Deglutition did not appear to be affected, and there was no pain.

The child remained under observation, and two months after admission, as the tumour had increased in size and was interfering with swallowing, a portion of the tongue, including the tumour, was removed with an écraseur, though it was afterwards found that not quite the whole of the tumour had been taken away. A fortnight after the operation recurrence *in situ* had been so rapid that the tumour was even larger than before. It was somewhat nodulated and very soft, and extended backwards as far as the finger could reach, feeling as if attached to the left anterior pillar of the fauces. It also encroached upon the floor of the mouth, and could be felt externally as a mass deeply situated in the left submaxillary region.

The patient was removed from the hospital, and died shortly afterwards. No autopsy was obtained. Microscopical examination of the tumour showed that "it was composed of delicate fine fibre-tissue, with cells exhibiting all the transitions in shape from round cells, through oval and spindle-shaped, to simple fibres."

Case 3 ('Guy's Surgical Reports,' 1873).—Charles J—, æt. 10 months, a healthy child with good family history. The mother first noticed a sore under the tongue at the age of seven months, which gradually developed into a "lump," and bled frequently. When admitted into the hospital under

Mr. Howse there was a white button-shaped eminence on the under surface of the tongue, extending from the floor of the mouth to the tip in the median line. It had a flattened surface, raised rounded edges, and a spongy appearance. After removal sarcomatous tissue was stated to exist in the base of the tumour.

The further history of this case is not known, as the patient did not return to the hospital.

Case 4 (Jacobi, 'Amer. Journ. of Obstetrics,' vol. ii, 1870, p. 81).—Thomas K—, æt. 11 weeks. He was born of healthy parents, and his brother (aged three years) showed no signs of syphilis. A few hours after birth the mother noticed a swelling of the tongue, but it did not interfere with suckling. It slowly enlarged, however, and was punctured, though nothing but blood was evacuated.

When the child first came under observation there was a tumour of the tongue the size of a walnut, occupying the left half of the tongue from the median line to the margin, and half an inch from the tip. It was well rounded inferiorly, encroaching much on the floor of the mouth, but the upper surface was less regular. It was moderately firm and elastic, of a bright red colour, and its surface was marked with a distinct network of large capillaries. The lymphatic glands were not enlarged, and in all other respects the child was healthy, though deglutition was now impeded owing to the increase in size of the swelling.

The tumour and the adjacent portions of the tongue were removed with a galvanic écraseur, and no hæmorrhage resulted. A fortnight later the wound was healing rapidly.

In the report of the microscopical examination the cells are described as mostly spindle-shaped, with oviform, oblong nuclei, and very uniform in size and shape; hence the tumour was considered to be a spindle-celled sarcoma. The external (? *peripheral*) portions of the tumour contained much muscular and interstitial tissue and capillaries, but less in proportion of the constituents of the growth proper.

No further history of this case has been published.

Case 5 (Bleything, 'New York Med. Journ.,' 1888, xlvii, p. 683).—A lad, æt. 17, had a painful "ulcer" of the tongue

on the right margin half an inch from the tip. It was covered with a small mass of granulations, and there was some induration of its base. The patient attributed the sore to a broken tooth. As repeated applications of the cautery failed to cure, and as the swelling enlarged and its base became harder, the affected portion of the tongue was removed; six years afterwards the patient was in excellent health, with no sign of recurrence.

The microscopical examination of the diseased tissue showed "areas of grouped cells, with new areas of infection around gorged vessels;" and after due consideration, and a not unnatural hesitancy between granulation and sarcomatous tissue, the tumour was declared to be a sarcoma of the tongue.

Case 6 (Godlee, 'Trans. Path. Soc.,' vol. xxxviii, p. 346, 1887).—A woman, æt. 24, came under observation with a swelling on the under surface of the left half of the tongue near the tip. It had only been noticed for five weeks. The tumour was hard, the size of a cobnut, and formed a slight projection upon the dorsum of the tongue; in the centre of the swelling there was a grey slough as large as a shilling. It was removed with scissors, and the wound healed rapidly. On inquiry it was ascertained that there was no recurrence of the disease three years after the operation.

Histologically the growth was an adeno-sarcoma. A large proportion of the tumour was made up of sarcomatous tissue, with round and spindle-shaped cells and many vessels. The glandular elements consisted of acini, varying much in size and shape. In the centre of the tumour was a small salivary calculus.

It was suggested that the tumour had probably arisen in connection with the mucous gland described by Blandin and Nuhn.

Case 7 (Heath, 'Trans. Path. Soc.,' vol. xx, p. 157, 1869).—The patient was a man, æt. 60. The disease had existed six months. On examination "there was a ragged ulcerated surface in the floor of the mouth between the tongue and the lower jaw on the left side. This extended to the side of the tongue, and with the finger a large mass could be felt in the

substance of the tongue, reaching beyond the median line, and to about two inches from the lips." There was constant pain in the tongue, but the patient's health was otherwise good. The lymphatic glands were not affected.

The anterior half of the tongue was removed freely. Twelve years afterwards the patient was in good health, and the stump of the tongue was sound.

The specimen on section presented the appearances of a medullary cancer. The report of the Morbid Growths Committee is as follows: "The tumour was for the most part embedded in the under and outer part of the left side of the tongue, not far from the tip, but a portion of it was prolonged downwards among the muscular attachments of the tongue, and not improbably involved a part of one of the salivary glands. The tumour was small, well circumscribed, had an opaque white colour, and on scraping yielded not so much a juice as abundant minute fragments of apparently solid tissue. The part of it embedded in the substance of the tongue was spongy; the lower part was homogeneous and solid. On microscopical examination, the cell-elements of which the tissue was composed were in some places more or less regularly round or oval; in others they were elongated, or presented one or more tail-like processes." The remainder of this lengthy report is too vague for the purposes of exact histology, but it may be noted that there is no mention of the presence of bird-nest formations, of epithelial ingrowths, or of a definite alveolar structure. A drawing which accompanies the report most nearly represents a small spindle-celled sarcoma. The committee expressed their opinion that "the tumour was essentially a cell-growth invading and displacing the normal tissues, the cell-growth forming equally the more obvious cell-structure of the tumour and its fibrous portion."

Case 8 (Albert, 'Wiener medizinische Presse,' 1885, p. 171). —The patient, a woman æt. 56, stated that she had suffered from difficulty in swallowing for nearly a year. A month after the onset of this symptom she noticed a swelling on the tongue, which slowly but steadily enlarged. On examination a smooth roundish tumour was found to be attached to the

root of the tongue, and it extended upwards almost to the hard palate, concealing the isthmus faucium. The attachment of the tumour involved the whole breadth of the tongue, to which it was firmly fixed, and its outline was well defined. Its consistency was moderately hard throughout, except in one or two spots which felt decidedly soft. Excision of the whole tongue was performed through the submaxillary region. Death took place on the eighth day after the operation from bronchitis and lobular pneumonia. The microscopical examination of the tumour showed that it was a round-celled sarcoma.

Case 9 (Poncet, 'Lyon Médical,' 1888, lviii, p. 95).—The report of this case states that the patient was a man, æt. 32, who came under observation for a tumour of the tongue which had been growing for eight years. The lower jaw was divided and the tongue excised. The tumour weighed 400 grammes, and was considered to be a fasciculated sarcoma originating in the substance of the tongue, very probably in the submucous and intermuscular cellular tissues.

In reply to an inquiry respecting the further history of this case, Prof. Poncet very kindly informed me that his patient was in excellent health fifteen months after the operation, with no sign of recurrence of the tumour. The publication of a more detailed account of this case is contemplated.

Case 10 (Hutchinson, 'Med.-Chir. Trans.,' vol. lxviii, p. 311).—The patient was a man, æt. 22. A swelling was first noticed on the left side of the tongue at the age of ten years. It continued to increase without causing inconvenience, except from its size, until he was twenty-one. The tumour consisted of a large rounded mass, deeply embedded in the tongue, and wedged in between the sides of the lower jaw. The tip of the tongue was free, and could be moved on the surface of the tumour, but with this exception the whole of the organ was involved. The mucous membrane of the posterior two thirds of the tongue on the left side was covered with a coarse papillary growth, continuous with the substance of the tumour beneath, and not ulcerated. A year later the growth had increased considerably, and the inconvenience

was unbearable. It was removed by division of the lower jaw and the application of an écraseur round the base of the tongue just in front of the epiglottis. A rapid recovery followed, and the patient continued well for many months; subsequently, however, the growth recurred in the floor of the mouth, and spread rapidly, causing death, partly by pressure and partly by hæmorrhage, two years and a half after the operation. There was no evidence of glandular enlargement. On examination the tumour was almost globular in shape, measuring two inches and a half in diameter, and not encapsuled. It weighed seven ounces. It completely replaced the substance of the tongue, save a small portion three fourths of an inch in length at its tip. The posterior part of the new growth showed a greyish fibrous structure, which was divided into loculi by white bands; in front it was softer and more vascular. The muscular tissue of the tongue was inseparably attached to the tumour. Histologically the growth was a round-celled or lympho-sarcoma, consisting of large rounded alveoli filled with lymphoid cells of uniform size, enclosed by bands of fibrous tissue and intersected by finer strands of the latter. Between the cells a delicate reticular tissue was seen in parts. Here and there in the substance of the tumour muscular fibres, atrophied by pressure, were observed.

Case 11 (Butlin, 'Lancet,' 1887, vol. i, p. 623).—A man about 40 years of age was admitted with a swelling of the left half of the tongue. Two months previously he had noticed a soreness of the tongue in the region described, but the swelling had only appeared a month before admission, and during that interval it had increased rapidly.

When first examined a smooth, soft, elastic swelling was found in the substance of the left half of the tongue. It was about the size of a racket-ball, and extended from the junction of the anterior and middle thirds to the root of the tongue, bulging on its upper and under surfaces, but not crossing the median line. There was no ulceration of the mucous membrane over it. The tongue could not be protruded from the mouth, but the patient complained of no pain except on eating. His general health was good. A hard enlarged gland was detected in the submaxillary region.

Large doses of iodide of potassium were administered, but as no diminution in the size of the tumour took place the left half of the tongue was excised three weeks after admission. The gland under the jaw was not removed. Five weeks later the patient was quite well, and could talk plainly. There was no glandular enlargement and no sign of recurrence.

At the present time, more than three years after the operation, the patient is in excellent health, and the condition of the mouth is very satisfactory.

On examination of the parts removed a soft white circumscribed tumour was found, with the appearance of a capsule at one spot. It was oval in shape, measuring one inch and a half in length by an inch in breadth, and was situated immediately beneath the mucous membrane of the middle third of the tongue on the left side. Microscopically it consisted of sarcoma-cells, which were large, round, and granular. The matrix was generally homogeneous, but in parts granular. Numerous small blood-vessels permeated the section. The tumour belonged to the class of lympho-sarcomata.

In addition to the above cases two examples of connective-tissue tumours of the tongue have been recorded by Eve ('Trans. Path. Soc.,' vol. xxxvii, p. 223), but as they have no clinical history, and differ widely in anatomical details from the tumours at present under consideration, no further mention of them need be made. Butlin ('Diseases of Tongue,' p. 259) refers to one case of tumour of the tongue, sections of which he had the opportunity of examining, but he did not see the tumour itself, and knew nothing of the clinical features of the case. "It presented the characters of a round-celled or lympho-sarcoma," he adds.

Barker ('Holmes' System of Surgery,' vol. ii, p. 576) mentions a case in which a somewhat pendulous growth upon the dorsum of the tongue was removed and soon recurred. Several growths of the same kind appeared subsequently on the skin of different parts of the body. He regarded the case as in fact an example of multiple sarcomata, the dorsum of the tongue participating in a general disease of the cutaneous surface of the body. Finally, a case is recorded by Santesson, of Stockholm, of an angeio-sarcoma of the tongue, but I can

give no further details, as the original description is inaccessible.

Having thus dealt with the literature of the subject as fully as possible, it only remains for me to review shortly the cases quoted above, and, in conclusion, to make a few remarks on their clinical and pathological bearings. Of the eleven cases described in more or less detail, Nos. 1, 2, 7, 8, 9, 10, and 11 have some points in common, and will be considered together. Case 5, though declared to be a sarcoma, was probably inflammatory, as it began in an irritation-ulcer; it was composed of "areas of grouped cells, with new areas of infection around gorged vessels;" and there were no signs of recurrence at the end of six years. Case 6 must be considered as an adeno-sarcoma of a mucous gland, rather than a primary tumour of the substance proper of the tongue, and should therefore be placed in a different category from that of the majority of the cases here recorded. Of Case 3 the evidence is insufficient to enable one to form an opinion as to its nature; it might have been an inflamed nævoid growth. Lastly, Case 4, though quoted by all writers on this subject, is too vague for classification, and, as the accuracy of the diagnosis has been doubted by such a competent authority as Mr. Butlin, it may be put on one side.

Of the remaining seven cases it may be said that the disease was of the substance of the tongue and not of the surface, though a prominent mass was eventually developed in several instances; that the mucous membrane covering the tumour was not ulcerated (with one exception), and therefore pain was absent; that the lymphatic glands were not affected in the cases in which this point is mentioned, or only in the later stages, as in Case 1, where the submaxillary swelling was probably in the main an extension of the growth; and that local recurrence after operation was rapid in only one out of the six cases (excluding No. 8), while in three others it had not taken place at the time of publication. These remarks will be made clearer by the following scheme:

Case.	Age.	Length of history.	Situation, &c.	Results.
I	65	1 year	In substance of left half towards root; one gland enlarged	Recurrence within one year.
II	2	4 months	Middle third towards dorsum; glands normal	Rapid local recurrence. Death.
VII	60	½ year	In substance towards root; glands normal	No recurrence at end of 12 years.
VIII	56	1 year	Whole breadth of root of tongue	Death a week after operation.
IX	32	8 years	In substance	No recurrence at end of 15 months.
X	22	12 years	Deeply embedded, involving whole organ; glands normal	Death from local recurrence 2½ years after operation.
XI	40	7 weeks	In substance, left half; glands normal	No recurrence at end of 3 years.

The cases are too few to enable any useful conclusions to be drawn from the age and sex of the patients; the duration of the disease also varies within wide limits. But with regard to the latter point it is worth while to note that Case 9, which existed eight years, was a fasciculated sarcoma, and therefore probably of much slower growth than the remaining cases; and in Case 10, of still longer duration, Mr. Hutchinson was of opinion that the disease had begun in a congenital mole or nævus, and only developed "morbid tendencies" at a much later date. The similarity in histological structure of the tumours is not a little remarkable, for with one, or perhaps two, exceptions they were of the round-celled or lympho-sarcomatous type. Of the two exceptions, one was a fasciculated or spindle-celled sarcoma, and the other (Case 2) was of the mixed-cell variety. This point is of importance in the question of diagnosis, and will be referred to subsequently.

In a recent publication[1] on malignant diseases of the tongue and lips, Esmarch states that in his experience "the majority of tumours which are commonly recognised as sarcoma and extirpated belong to the syphilomata, and can

[1] Abstracted in 'Centralblatt für Chirurgie,' 1889, No. 29.

be cured by antisyphilitic treatment, whilst without this they frequently pursue a malignant course." The conclusions he draws are that the sarcomata of muscle-tissue are mostly syphilitic in nature (syphilomata); that as the tongue consists chiefly of muscular tissue, so syphilomata appear in it frequently, and that in many cases the origin of tumours (particularly sarcoma) is dependent upon a predisposition inherited from syphilitic ancestors. This theory derives little support from the cases here recorded, partly from the imperfection of the clinical accounts, and partly from the fact that in two of the cases (1 and 11) antisyphilitic treatment failed. Apart from the minute structure of the tumours, the fact of their recurring locally, and sometimes rapidly, is, I venture to think, opposed to such a view. Deep gummata of the tongue, with which sarcomata are most likely to be confounded, are said to be often multiple and of long duration, and will probably be associated with other syphilitic lesions, scars, ulcers, enlarged glands, &c. Admitting, then, the necessity of caution in the diagnosis of tumours of the substance of the tongue, I cannot at the same time doubt that malignant growths, which would be recognised by the majority of pathologists as sarcomata, do actually occur in the tongue, and stand in need of operative treatment. But whether they are due to ancestral syphilis, or represent a malignant transformation of an old syphilitic lesion, there are not enough facts at present to decide.

The difficulties of diagnosis, however, are not confined to syphilis. Chronic abscess and actinomycosis, and some innocent tumours too, will present swellings which must be distinguished from sarcomata. With regard to actinomycosis there are at least four cases of primary disease on record.

Ullmann[1] relates two such, and Hochenegg[2] and Hacker[3] one each. These cases presented nodules in the substance of the tongue, near its tip or borders, which were at first hard, but softened as they enlarged, and caused no pain. In one instance the tumour was the size of a pigeon's egg. The mucous membrane was not affected, but the submaxillary

[1] 'Wiener medizinische Presse,' 1888, No. 50.

[2] Ibid., 1887, No. 16.

[3] 'Wiener medizinische Wochenschrift,' 1885, No. 17.

lymphatic glands were enlarged. The source of infection could not be determined in all. The diagnosis appears to have been settled by puncture and microscopical examination of the contents. Though actinomycosis of the tongue has long been known in the lower animals, its occurrence as a primary disease in the human tongue is not yet generally recognised.

The treatment of sarcoma of the tongue scarcely calls for comment. Slow recurrence after free removal by operation is not peculiar to this region. But the preponderance of the round-cell type of growth in the examples quoted suggests the importance of an early and free excision.

In conclusion, I submit that there is a true bill for sarcoma of the tongue, but that it is a very rare disease as compared with epithelioma of that part, there being only four specimens preserved in the various London museums. The difficulties in its diagnosis are considerable, so that an adequate anti-syphilitic treatment should first be adopted in all cases. The resemblance which primary actinomycosis bears to it should not be forgotten, though operative treatment is required for both. In structure it is most frequently a round-celled growth, beginning in the substance of the tongue, the cause of which has yet to be determined.

Note.—Dr. Waller, of Rugby, writes to say that at the present time (August, 1890), nearly two years after the operation, the patient, Joseph P—, is enjoying fair health. There are large masses of secondary growth on both sides of the neck, also below the right zygoma and in the left sub-maxillary region, but there appears to be no recurrence in the mouth.

AN ANALYSIS

OF THE

CASES OF CHOREA

WHICH HAVE OCCURRED IN THE HOSPITAL DURING ELEVEN CONSECUTIVE YEARS.

By E. W. GOODALL, M.D.

In the first volume of the second edition of Fagge's 'Principles and Practice of Medicine,' on p. 753, will be found an analysis of cases of chorea occurring in Guy's Hospital for several years ending with the year 1878. In the present paper I bring the record up to the end of the year 1889, following the lines laid down by Dr. Pye-Smith in an article in ser. iii, vol. xix (1874) of these reports.

During the years 1879 to 1889 there were admitted into the hospital 262 patients suffering from chorea.

Sex.—Of these patients 74 were males and 188 females, which gives a proportion of 2 males to 5 females. Ten of the males and 17 of the females were admitted more than once, so that the number of *admissions* for chorea during these years was 298.

Age.—The first attacks were at the following ages :

	Male.	Female.	
At 3 years old	0	1	1
4 „	2	1	3
5 „	4	6	10
6 „	5	9	14
7 „	8	13	21
8 „	4	10	14
9 „	5	12	17
10 „	14	20	34
11 „	7	23	30
12 „	5	18	23
13 „	2	10	12
14 „	5	9	14
15 „	2	8	10
At 16 years old	1	10	11
17 „	1	8	9
18 „	2	11	13
19 „	2	5	7
20 „	0	2	2
21 „	0	1	1
22 „	0	2	2
24 „	0	1	1
27 „	1	1	2
30 „	1	0	1
34 „	1	0	1
36 „	1	0	1
	73	181	254

In 8 cases it is not stated at what age the first attack occurred.

From this table it will be seen that in this series of cases the first attack was found most frequently at from the fifth to the fourteenth year in males, and from the fifth to the nineteenth in females.

The series happens to contain 4 cases in which the disease appeared for the first time in males aged 27 years and upwards, an unusual circumstance, and worthy of note. The report of the patient aged 36 is headed "chorea," and reads exactly like that of a case of chorea; but the movements had commenced six months after an injury to the spine, and had not ceased at the end of eight months from their onset; they affected the muscles of the extremities, face, neck, and trunk. There was no history of acute rheumatism nor was there any cardiac murmur.

The patient who was first attacked by chorea at the age of 34 came into the hospital at the age of 37. The choreic movements had never left him since their first appearance three years previously. The man could assign no cause for their occurrence; he had never suffered from rheumatic fever, nor was he aware that any member of his family had been afflicted with either that disease or with chorea; he had been an inmate of various London hospitals on account of his misfortune.

When he first came into Guy's the diagnosis of his case was doubtful, chiefly on account of his age; but he had been in the hospital only eleven days when he had a well-marked attack

of acute rheumatism, during which a systolic apical murmur appeared. This attack was followed by two relapses. The patient went out at the end of nine months slightly relieved of the chorea, the murmur still being present.

The man in whom chorea first manifested itself in his thirtieth year was an inmate of Guy's nearly four years after its onset. In his case the movements had appeared immediately after an attack of acute rheumatism, and had become chronic. There was no heart disease. The family history is somewhat remarkable : it was stated that the patient's mother had been affected with chorea from her thirtieth year till her death at the age of 64; two of her sisters also suffered from chorea, which made its appearance after they reached maturity, and in one of them certainly the disease became chronic.

As regards the male patient aged 27, in his case also the chorea followed close upon an attack of rheumatic fever. Mitral regurgitation was diagnosed and aortic endocarditis suspected.

The case of the woman aged 27 will be related amongst the fatal cases.

Recurrence.—Of the 262 patients 104 suffered from more than one attack of chorea (39·6 per cent.); one female aged 23 had had seven attacks since she was 16; another seven during nine years (8 to 16); two others six in nine (3 to 11) and ten (7 to 16) years respectively; and two girls five in five years (3 to 7 and 7 to 12). Eight of the cases had become chronic: two (males) have already been mentioned; the rest are as follows :—F., 22, chronic since her tenth year; F., 13, since her eleventh; M., 18 (who died), since his seventh; M., 12, since his fourth; F., 28, since her eighteenth; this woman was also the subject of Graves's disease, the symptoms of which first showed themselves during her twenty-fifth year. None of these chronic cases were cured, though some were much relieved.

Season.—As attention has been directed[1] to the relation of chorea to season, I have drawn up a table of 258 cases in which it was stated in or about what month the attack of chorea commenced :

[1] Gowers, 'Diseases of the Nervous System,' vol. ii, p. 548.

Jan.	Feb.	March.	April.	May.	June.	July.	Aug.	Sept.	Oct.	Nov.	Dec.
22	26	20	25	25	21	15	13	13	20	23	35

—that is in the first quarter, 68; in the second, 71; in the third, 41; and in the fourth, 78.

Distribution.—There were 60 cases of hemichorea, reckoning by the number of admissions; of these, 25 were right-sided and 22 left. In 7 more cases the movements, being at first confined to the right side, became afterwards left-sided also; while similarly in 6 other cases they were at first left-sided. Among these 60 cases are included two in which the patients were twice in the hospital for hemichorea; one had left hemichorea on both occasions, and the other had left hemichorea the first time and right the second. Of the 60 cases 13 were males and 47 females (of whom two were admitted twice).

Condition of the heart (excluding fatal cases).—In 65 patients there was a systolic murmur more or less limited to the apex, and in some cases it was temporary; of these 20 had rheumatic fever either previously, or at the time the murmur was noted; while of the rest one had acute nephritis, and another had been in the hospital with chlorosis shortly before the attack of chorea, the same murmur having then been present. In none of these 65 patients was mitral regurgitation thought to exist. But in 33 other patients reflux through the mitral valve was diagnosed, and of these 22 had had rheumatic fever. Mitral incompetence together with pericarditis was found in 4 other cases, of whom 3 suffered from acute rheumatism. Stenosis of the mitral valve (with or without regurgitation) was present in 13 patients, but of these only two had had rheumatic fever. In one patient there was aortic stenosis, which was known to have existed before the chorea came on; there was no history of rheumatism. Two patients had aortic stenosis with regurgitation; both had had rheumatic fever; another suffered from the same kind of valvular disease with mitral regurgitation in addition; while still another presented the signs of aortic stenosis and regurgitation, mitral regurgitation and pericarditis; in both of these cases there was rheumatic fever. Of 4 cases in which pericarditis without any valvular disease was diagnosed, 3 had rheumatic fever. In 10 cases there was a systolic basic murmur, noted as probably hæmic; of these there was

a history of acute rheumatism in only one. In one patient who had previously suffered from rheumatic fever there was a loud systolic murmur heard with equal intensity all over the cardiac area. In only one case is no mention made of the condition of the heart.

There were 11 deaths; excluding these we have 250 cases in which the condition of the heart was noted; in 115 of these the heart was normal, and of these 21 had rheumatic fever; in 135 a bruit of some kind or other was detected, and of these 56 underwent an attack of acute rheumatism either previously to or simultaneously with the chorea. It will have been observed that, with the exception of the cases of mitral stenosis, it was especially in those patients who were the subjects of a serious cardiac lesion that rheumatic fever was found; and apart from chorea we not infrequently meet with cases of mitral constriction in which no history of rheumatism can be obtained. There were 28 cases of first attacks of chorea in which were present symptoms pointing to heart disease of longer standing than the chorea; in 16 of these there was a history of previous acute rheumatism; in 12 there was none.

Of the 115 patients whose hearts presented no abnormality, in the cases of only 21 was it stated that rheumatic fever had previously occurred, 3 of them being perhaps a little doubtful. It is of course not unlikely that in some instances the reporter has omitted to accurately ascertain the previous occurrence of rheumatic fever; but in most of the reports it seems to be but seldom that no inquiry at all has been made into this point.

Fatal cases.—1. F., 12, second attack of chorea, left-sided; rheumatic fever two years before, followed by chorea. Brain normal to the naked eye; hypertrophied and dilated heart; mitral valve incompetent, with a fringe of granulations along its edge; passive effusion into the pericardium; viscera congested.

2. F., 19, first attack; rheumatic fever three months previously; a second attack of rheumatic fever soon after she came into the hospital. Recent vegetations on the mitral and aortic valves; pulmonary embolism, the clot having been detached from a thrombosed right femoral vein; broncho-pneumonia; brain normal to naked eye.

3. M., 8; second attack; first six months before, after acute rheumatism. Pericardium slightly adherent; heart very large, 13 oz.; mitral valve incompetent, thickened and fringed with granulations; so also were the aortic sigmoids, which in addition were a little everted.

4. F., 11; second attack; first five years before, after a slight attack of rheumatic fever. Pericarditis; incompetent mitral valve with recent vegetations. There was also gangrenous stomatitis with phlebitis of the left innominate vein, mediastinitis, and left empyema.

5. M., 19; second attack; first when he was a little boy; acute rheumatism two or three months before the second attack of chorea; patient became maniacal before his death. Recent vegetations were found upon the mitral valve, with ulceration at the junction of the valve with the auricle. Brain and cord examined microscopically and found quite normal.

6. F., 17; second attack; first at nine years of age; never had rheumatic fever; a pericardial rub was heard; patient died suddenly and unexpectedly. Brain normal to naked eye; slight pericarditis; recent endocarditis of aortic and mitral valves; kidneys, 13 oz. the pair, enlarged and red; cortex swollen; in a state of acute nephritis.

7. M., 18; had suffered from chorea, off and on, since he was seven years old; had twice before been in Guy's with it; had rheumatic fever some years ago, and again just before his death. There was mitral stenosis and ulcerative endocarditis of the mitral and tricuspid valves; embolism of the left posterior cerebral and common iliac arteries, the latter leading to gangrene of the foot; infarction of spleen and kidneys.

8. F., 27; first attack; rheumatic fever four years before; the chorea was confined to the left side when the patient was admitted, but afterwards became universal; she became maniacal before death. The mitral valve was thickened, and evidently allowed regurgitation; it also presented a row of vegetations along its edges; infarcts in the kidneys.

9. F., 7; third attack; first one year, second six months, before; report does not state whether there had been rheumatic fever or not. Pericardium adherent; mitral valve some-

what narrowed; vegetations on the edges of the aortic and mitral valves; left auricle distended.

10. M., 8; first attack; patient was convalescent from a first attack of acute rheumatism. Acute pericarditis; mitral valve incompetent, one segment being much shortened; recent vegetations upon it and also upon the aortic valve; hypertrophy of left side of heart; viscera as in cases of mitral regurgitation.

11. F., 13; second attack; was in Guy's for the first attack nearly eighteen months before; had an attack of rheumatic fever closely preceding the first attack of chorea; she then had pericarditis and mitral regurgitation; she had another attack of acute rheumatism, with a second attack of chorea. The pericardium was adherent; the flaps of the mitral valve shrunken and fringed with recent vegetations.

Thus it will be seen that in all these fatal cases there was recent endocarditis of the mitral valve, while in 5 the aortic valve was also affected. In two cases the endocarditis was of the ulcerative variety. Nine of the patients had certainly suffered from acute rheumatism; 1 certainly had not; in the remaining case the report does not say one way or the other.[1]

Relation to rheumatism. — Of the 251 patients, 71 undoubtedly underwent an attack of rheumatic fever at some period or other; and in 6 other cases it was extremely probable from the history obtained that there had been a previous mild attack of the same disorder.[2] In the large majority, 59, of these cases the rheumatism preceded the chorea; in 34 of the 59 the chorea came on within a few days or weeks of the rheumatic attack, while in the remaining 25 there was an interval of months or years between the first attack of rheumatism and the chorea, whether accompanied by another attack of rheumatism or not. In 16 cases the chorea preceded

[1] Besides these fatal cases may be mentioned that of a little boy, aged 7 years, who was admitted into Guy's with a first attack of chorea in the winter of 1882. He had never had rheumatic fever, but he was found to be affected with mitral stenosis and regurgitation. He was an in-patient with a second attack of chorea in the spring of 1884. From both of these attacks he recovered; but he died of the mitral disease in the hospital in July, 1886. He never had rheumatic fever.

[2] These six cases have been included amongst the rheumatic cases in the paragraph headed "Condition of the heart," above.

the first attack of rheumatism by a period of months or years but in not a single case was a first attack of chorea closely followed by a first attack of rheumatic fever. In 2 cases it was not ascertained exactly which of the two diseases was the first to manifest itself.

These 77 cases are exclusive of those which proved fatal; if these be added, then out of 262 patients 80 certainly and 6 most probably had rheumatic fever (32·8 per cent.).

There was a distinct family history of "rheumatic fever" or "acute rheumatism" in 43 instances (excluding cases in which it is merely stated that the father, mother, or some relation was "rheumatic," or had "suffered from rheumatism"). In 17 of these it was the mother, in 10 the father who was so affected, and in 3 both parents.

In 28 instances there was a family history of chorea; in 8 it was the patient's mother who was so affected, in 16 a brother or sister; in no instance is it stated that the father had ever had chorea.

In 8 cases there was a history of both acute rheumatism and chorea in the family, the most striking examples being those of two girls, of whom one was attacked by chorea and rheumatic fever, her father and mother having suffered from rheumatic fever, a brother from rheumatic fever and chorea, another brother having died from chorea; while the other had chorea twice, but never rheumatic fever, her mother, two of her sisters, and two of her brothers having suffered from chorea, and one of the latter from rheumatic fever as well.

Causation (apart from rheumatism).—In 42 instances fright was assigned as the cause of the chorea; in only 5 of these was there rheumatic fever.

In 3 cases there was distinct evidence of the disease having arisen through imitation of another choreic patient, and 2 of these had rheumatic fever.

In 8 cases the chorea came on very soon after a fall or head injury; in one of these acute rheumatism was present.

There were 10 examples of chorea occurring during pregnancy; in 7 of these the patients were married:

1. Æt. 20; third pregnancy, third month; second attack of chorea; first after last confinement; no rheumatic fever; no bruit.

2. Æt. 22; married three years, second pregnancy; first attack of chorea; rheumatic fever fifteen months before; no bruit.

3. Æt. 22; seventh month of third pregnancy; married five years; first attack of chorea; no rheumatic fever; pulmonary systolic bruit.

4. Æt. 22; fourth month of first pregnancy; married two years; second attack of chorea; first ten years before caused by fright; rheumatic fever six years ago; apical systolic bruit.

5. Æt. 23; fourth month of first pregnancy; several previous attacks of chorea; first nine years ago, due to fright; no rheumatic fever; no bruit.

6. Æt. 24; third or fourth month of first pregnancy; first attack of chorea; no rheumatic fever; no bruit.

7. Æt. 25; sixth month of fifth pregnancy; third attack of chorea; first seven years ago (before marriage), caused by fright; second four years ago during first pregnancy; no rheumatic fever; no bruit.

Of the remaining three cases, in only one does the report state that the patient was unmarried; in another (No. 9) it says that the patient "went out on account of the pregnancy being found out by other women in the ward," so that she probably was not married. In the third case (No. 8) no statement one way or the other is made.

8. Æt. 18; fifth month of ? what pregnancy; second attack of chorea; first, four years before. No rheumatic fever; no bruit.

9. Æt. 19; fifth month of ? what pregnancy; fifth attack of chorea; first, ten years ago; rheumatic fever twelve years ago; has mitral regurgitation.

10. Æt. 21; about seventh month of ? what pregnancy; first attack of chorea; no rheumatic fever; apical systolic bruit.

A CASE

OF

FRACTURE OF THE CORACOID PROCESS

UNCOMPLICATED WITH OTHER INJURY.

WITH A STUDY OF THE LESION.

By R. CLEMENT LUCAS, B.S.

I. *Clinical Account of a Case in which there was Wide Displacement of Fragments.*
II. *Five Modes of Fracture.*
III. *Forms of Injury.*
IV. *Mode of Union.*

The rarity of fracture of the coracoid process has limited the study of the injury to few observers, and the reports of cases are scanty; it behoves one, therefore, who has an exceptional opportunity, to make as careful and exact an observation as his means allow. I make no other apology, should it appear to any that this is a lengthy description of a rather trivial injury.

On October 4th, 1889, Mr. Devenish Meares, the house surgeon in charge of the surgery, asked my assistance in determining the nature of an injury to the shoulder. The patient, R. H—, was a man æt. 58, and the accident had

occurred about twenty-four hours before he sought advice. About 6.30 in the evening of October 3rd he was employed in piling up a number of market baskets, and when the pile was about seven feet in height he slipped off and fell forward on his shoulder. He did not suffer much pain at the time, but experienced numbness of the arm and hand with loss of power. He had to stop working as a consequence. He came to the surgery for advice about four o'clock on the following day. There was a little effusion about the front of the shoulder, but very slight superficial bruising. Mr. Meares had discovered a small loose piece of bone lying beneath the anterior edge of the deltoid muscle and three inches below the clavicle. It was easy, by a process of exclusion merely, to prove this could be nothing but the coracoid process. It could not be part of the head of the humerus, for this moved smoothly and without pain in its glenoid cavity; it was not the great tuberosity, or part of it, as that would be drawn upwards and outwards by its muscles; it was not the lesser tuberosity, for that would be drawn inwards by the subscapularis; the acromion and clavicle could be felt intact: it followed, therefore, that this must be a separated coracoid process. We, however, by more direct methods showed that the loose piece of bone was the fractured and displaced coracoid process. On feeling in the usual situation for that process just below the clavicle, the man flinched immediately the finger touched the spot, but I thought I could still detect the base of the process through the fluid blood-swelling, though it was evidently less prominent than on the other side. The patient also complained of pain when the loose piece of bone was moved. We measured with a tape the distance the bone was displaced, and we found it measured two inches and three quarters from the base of the coracoid, the displacement being *downwards* and slightly *outwards*. We next, by watching the effect of muscular action, proved conclusively that the loose bone was the coracoid process acted upon by the short head of the biceps and the coraco-brachialis, but unattached to the pectoralis minor. When the arm and forearm were fully extended and abducted the point of bone descended lowest, being then fully three inches from the clavicle. On flexing fully the forearm, adducting the arm, and drawing the elbow

across the chest, the piece of bone could be raised nearly to its proper position, and Mr. Meares thought he could detect indistinct bone crepitus, though I could not satisfy myself that what I felt was more than the crepitus of effusion. When the biceps was thrown into action by voluntary flexion of the forearm the piece of bone again descended. The patient's power of flexion was feeble, so that a one-pound weight placed on his palm altogether counterbalanced the power of bending his arm. It was evident that the flexion was mainly carried out by the long head of the biceps muscle.

There had been no previous injury to the shoulder, and there were no signs or history of osteo-arthritis or rheumatism.

The treatment adopted was to flex strongly the forearm, bring the arm to the chest-wall and the elbow forwards. In this way the fragment was brought into apparently good approximation to the base of the process. The arm was secured by means of strapping to the thorax, and a pad was placed over the acromial end of the clavicle.

I regret to say I cannot state with certainty the ultimate result of the treatment, for after a fortnight the patient ceased to attend, and a letter sent to his address failed to elicit a reply. He appeared to be intolerant of the confinement necessary to keep the fragment in position, and to aim at loosening his bandages. It may be concluded that only fibrous union, under the circumstances, could possibly have taken place.

The possibility of wide separation of the fragments, of which this case is an illustration, appears not to have been generally recognised as having occurred in clinical experience, and Hamilton goes so far as to deny the chance of its occurrence. In making a very natural protest against the descriptions of deformities deduced without clinical knowledge from the supposed action of muscles on fractured ends of bone, he overstates the case and falls into error. His remarks are as follows :—"It has been generally stated that when this process is broken off it will be carried downwards by the united action of the pectoralis minor, the short head of the biceps, and the coraco-brachialis muscles ; but this will depend upon whether the coraco-clavicular ligaments are ruptured also—a

circumstance which is not very likely to occur, at least to any great extent; and, in fact, *not one of the well-attested examples of this fracture has ever been accompanied with any considerable displacement in this direction.*"[1]

I will now proceed to the consideration of the various points suggested by the foregoing case, and endeavour to obtain additional light from cases previously reported.

Modes of Fracture.—There would appear to be as many as five ways in which the coracoid process may be fractured. 1. By direct violence. 2. By dislocation upwards and forwards of the head of the humerus. 3. By extreme flexion of the shoulder-joint, when the coracoid process is thrown into forcible contact with the under surface of the clavicle. 4. By downward crushing of the clavicle upon it. 5. By sudden muscular action.

We will consider the evidence in favour of the various ways in which the fracture may be brought about.

1. *By direct violence.*—This is the way generally described as usual, and by the writers of many of our text-books as the only way in which the process can be fractured. Hulke,[2] in Holmes's 'System,' speaks of it as "an extremely rare accident, and only produced by severe direct violence." Erichsen[3] is equally dogmatic. "It cannot happen," he says, "except by very direct violence."

Bryant[4] quotes two cases caused by direct violence, to which I shall again allude. Hamilton[5] speaks of two cases which came under his own observation, both of which were complicated with dislocation of the acromial end of the clavicle. One of these accidents occurred to a man who was struck by a board which fell edgewise on his shoulder; the other happened to a girl, æt. 17, as the result of a fall on her shoulder. In my own case there was evidence of direct violence, and the biceps being tense at the time was the cause of the great displacement.

[1] Hamilton, 'Fractures and Dislocations,' 7th edit., p. 249.
[2] 'A System of Surgery,' 3rd edit., vol. iii, p. 953.
[3] 'Science and Art of Surgery,' 8th edit., by Beck, vol. i, p. 566.
[4] 'Practice of Surgery,' 4th edit., vol. ii, p. 418.
[5] 'Fractures and Dislocations,' 7th edit., p. 248.

2. *By dislocation upwards and forwards of the head of the humerus.*—The fracture may be brought about in this way, whether the dislocation be partial or complete. The first case of the kind published was by South,[1] and in his case the dislocation was incomplete, the head having been proved by dissection not to have left its capsule. The report I will give in a condensed form.

A painter, æt. 58, fell thirty feet, was brought to the hospital perfectly sensible, but with contusions on head and severe injury to his left arm. The olecranon process was broken and the elbow-joint laid open by a wound behind the joint. The roundness of the shoulder on the same side was not lost, but a depression was observed below and behind the acromion. On gently rotating the arm the bone slipped into place, but again slipped out whilst the elbow was being adjusted. When replaced a second time grating was heard. The man died on the fifth day, having suffered from vomiting, cough, and pain in the pit of the stomach. Only the shoulder was examined; the clavicle and acromion were both found broken but not displaced, the latter at one inch from its extremity, the former at the junction of the middle and outer thirds. "The coracoid process was found broken, about half an inch from its tip, into two unequal pieces, the smaller of which remained connected above with a piece of the triangular ligament still attached to the acromion, and below to the short head of the biceps muscle, which had pulled it down as far as the ligament would allow, about half an inch below and to the outer side of the stump of the coracoid process. This muscle was torn from the coraco-brachialis about an inch; and to the top of the conjoined tendon of the latter and of the lesser pectoral muscle was attached the larger portion of the broken coracoid process, which had been drawn rather lower than the other portion, and to the inner side, but it was still connected by a thin cord of fibrous matter to the coracoid process. In front of the capsule of the joint was a slit about an inch in length, through which the cartilaginous covering of the head of the humerus was seen glistening." This rent was not large enough to allow the escape of the head, and the partial dislocation for-

[1] 'Med.-Chir. Trans.,' vol. xxii, p. 100.

wards, or upwards and forwards, was made artificially after death.

In the case reported by Holmes[1] the manner in which the injury was caused is similar to that which occurred in South's, but the dislocation of the humerus was complete in a direction upwards and forwards.

A man, æt. 50, fell thirty feet, striking the left elbow, chest, and head, and was admitted into hospital with concussion. The elbow-joint was extensively laid open, the end of the ulna being broken into numerous pieces, several of which were removed. In front of the outer part of the clavicle the head of the humerus formed a rounded swelling; the arm was shortened and the elbow away from the side. There was crepitus on rotation and difficulty in reduction, which led Mr. Tatum (under whose care the case was admitted) to diagnose fracture of the coracoid process. Insensibility continued for three days, and then signs of septicæmia occurred, and the man died of pyæmia on the fourteenth day with the dislocation unreduced. "The head of the humerus was found immediately under the skin, having passed through the fibres of the deltoid muscle, and leaving the cephalic vein on the inner side. It had fractured the coracoid process in its passage upwards, and was resting behind on the stump of this process, and on the clavicle, with a small portion of the coraco-acromial ligament, which remained untorn. *Internal* to it (besides the fibres of the deltoid and the cephalic vein) was found the fractured extremity of the coracoid process, with the muscles attached to it, viz. the pectoralis minor, short head of the biceps, and coraco-brachialis. *External* and somewhat posterior to it was the acromion process, separated from it by some of the fibres of the deltoid. *Below* and a little external to it was the glenoid cavity, the tip of which lay on a horizontal plane quite below the level of the dislocated head of the bone. The long tendon of the biceps remained still attached to the scapula, and was therefore situated below and external to the head of the humerus. The bone in passing out of the glenoid cavity had injured this tendon slightly, so that some of its internal fibres had been broken away from the muscle, and remained floating freely

[1] 'Med.-Chir. Trans.,' vol. xli, p. 447.

with a tuft of muscular fibres attached to them. The coracoid process had been fractured near its base. The coraco-acromial ligament remained attached to both fragments, so as to prevent their being separated to any great extent. The tip was drawn a little downwards and inwards by the muscles attached to it. The muscles attached to the greater tuberosity of the humerus were torn through, except that a portion of the teres minor remained. The capsular ligament had been ruptured at its upper and inner part, forming a large hole for the passage of the head of the humerus."

It will be noticed upon examining these two reports that the injury at the shoulder was in each case secondary to a fall upon the elbow, for the olecranon was in both cases broken and the elbow-joint laid open. In South's case the force transmitted through the shaft of the humerus to its head, after splitting the coracoid process into three portions, expended itself upon the clavicle and acromion, causing fracture without displacement of these bones. In Holmes's case the coracoid process was broken near its base, and the head of the bone then slipped forwards and upwards in front of the clavicle and acromion through the fibres of the deltoid muscle.

3. *By extreme flexion of the shoulder, when the coracoid process is thrown into forcible contact with the under surface of the clavicle.*—In a paper read before the Royal Medical and Chirurgical Society in 1887, Mr. Arbuthnot Lane[1] drew attention to the fact that in extreme flexion of the shoulder the scapula continued to rotate until obstructed by contact of the coracoid process with the under surface of the clavicle, and he proposed that this anatomical fact should be made use of to enable a surgeon to break down adhesions in the shoulder-joint. He further suggested that it was not improbable that the coracoid process might be fractured in this way, and in illustration alluded to the two cases quoted by Hulke in Holmes's 'System of Surgery' as probably caused in this manner. They are thus described :—"Two cases of fracture of the coracoid process have come under my notice. In both the fracture was caused by a fall forward from a slight height with the arms stretched forward; there was mobility of the tip of the process, with crepitus and pain, but

[1] 'Brit. Med. Journ.,' Nov. 12th, 1887.

not any displacement."[1] If this account be correct, Mr. Lane's explanation would appear to be the only possible one. There is an apparent contradiction between the cases quoted and the statement made a few lines before to the effect that it is an accident "only produced by severe direct violence."

4. *By downward crushing of the clavicle upon the process.*—An instance of this mode of fracture is related by Dr. Kelly[2] in the Pathological Society's 'Transactions.'

"George G—, æt. 33, a carman, was driving a waggon down Holborn Hill in April, 1868, when he slipped, and one of the wheels passed over his left shoulder and head, causing extensive fracture of the skull and instantaneous death. At the post-mortem examination it was found that the scapula was broken in half transversely about an inch below the spine, and also at the superior angle. The acromion process was fractured obliquely from within outwards; the coracoid process was broken off about three quarters of an inch from its outer extremity. The second and third ribs on the left side were broken, but the humerus and clavicle were uninjured. There was, however, a slight graze on the articular surface of the humerus, close to the bicipital groove, caused by the pressure of the acromion upon it. The acromion process, when fractured, would allow the weight of the waggon to press through the clavicle upon the coracoid process."

To this mode of fracture must also be referred a case which occurred in the practice of Arnott.[3] The case, however, was, correctly speaking, a separation of the epiphysis.

"E—, a boy fifteen years of age, was brought to hospital September 16th, 1838, having fallen from a tree. He had fracture of the skull, requiring the use of a trephine; the left clavicle was also broken, and two of the ribs of the same side. He lived for ten days. On the post-mortem examination, besides the injuries already mentioned, it was found that the coracoid process of the scapula had been broken off at its root. The point of fracture was just where the process becomes joined as an epiphysis above the glenoid surface to the body of the bone; and, considering the age of the patient,

[1] 'System of Surgery,' 3rd edit., vol. iii, p. 953.

[2] 'Path. Soc. Trans.,' vol. xx, p. 270.

[3] 'Med.-Chir. Trans.,' vol. xxii, p. 109.

there can be little doubt that perfect union had not been established. Owing to the insensibility of the boy, and his constant motion, resulting from the injury of the head, it was not possible to keep the arm at rest or properly confined in its bandages; consequently an abscess had formed around the fractured ends of the clavicle, and also between the fractured coracoid process and its corresponding surface."

I think there can be little doubt that this case of separation of the epiphysis should be placed in the same category with Dr. Kelly's, as caused by the pressure of the clavicle upon the process. The severe injury to the head and the fractured clavicle indicate that the impetus of the fall had been received on the shoulder and head.

5. *By sudden muscular action.*—A case of fracture of the coracoid process by muscular action has been described by Dr. Edward Hulme as occurring in the Dunedin Hospital.[1]

T. H—, æt. 57, a miner. "Three weeks previously he was on a bank in the act of passing through a wire fence when he slipped, and in falling his left arm caught in one of the wires. He instantly felt a severe pain in the fingers, followed by loss of power in the arm and inability to raise the elbow from his side. On examination it was found that the coracoid process of the left scapula was fractured and drawn downwards." The case was treated by flexion and bandaging of the arm to the side, and a flat piece of cork over the process. "After six weeks the fracture was firmly united, but with the end of the process somewhat depressed."

Forms of Injury.—From the reported cases one must assume that there are two forms in which fracture of the coracoid process may occur; and these two forms correspond to the two varieties met with in other situations, where a process of bone having strong muscles connected with it has, in addition, a support from ligament or tendinous expansion. I think I have shown that just as with the patella and olecranon process there are two forms of fracture, in one of which there is wide separation of the fragments and in the other little separation, so in the coracoid process these two forms of fracture may be met with. A glance at the anatomy of the pro-

[1] 'Lancet,' Nov. 22nd, 1873, p. 737.

cess will show the probability of this apart from clinical observation. The strong conoid ligament attached to its angle is continued forwards on its inner margin and upper surface as trapezoid. The pectoralis minor connected with the same border sends an expansion over the anterior part of the upper surface, whilst the postero-external border is occupied by the coraco-acromial ligament, and the tip is grasped by the common tendon of the coraco-brachialis and short head of the biceps. In addition to these structures we have the transverse and the coraco-humeral ligaments attached to the margins of its base. Being thus surrounded with ligament and tendon, we must expect to meet with a certain number of cases in which when fractured the process shows little tendency to displacement.

To this first variety, which corresponds to fracture of the patella or olecranon by direct violence, must belong the two cases mentioned by Bryant:[1]—"I have seen but one decided instance, and that in a girl, æt. 15 or 16, the result of a blow. There was local pain and crepitus to denote the injury, with displacement of the process, the point of which was drawn downwards by the biceps muscle, and its base projecting. Dr. Pauli, of Luton, kindly gave me the notes of a case that occurred in a girl, æt. 14, from a fall on the shoulder. There was marked mobility of the coracoid process and crepitus, but no deformity. There was pain in the part, increased by any movement of the arm."

It is evident that as mobility and crepitus were the two symptoms by which these cases were diagnosed, there could not have been any great displacement. Judging by the time the coracoid epiphysis unites, these cases ought, however, to be classed as separations of the epiphysis. Now as ossification in the epiphysis is nearly completed by fifteen or sixteen, the line of separation would be near the angle, a part well within the attachment of strong ligaments; and the muscles at this age, especially in girls, would be scarcely of a power to tear through the ligaments holding the fragments together.

The two chief forms of injury are (1) where the fragments are held in contact by ligaments and tendinous expansion, and

[1] 'Practice of Surgery,' 4th edit., vol. ii, p. 418.

(2) where there is wide separation by muscular action. To these may be added (3) where the process is comminuted as in South's case, and lastly (4) where there is a separation of the epiphysis.

The case I have related belongs to the second variety, in which the action of the muscles attached gives rise to wide separation of the fragments, a variety which corresponds to the common displacement met with in transverse fracture of the patella or olecranon. We were careful to measure the displacement with exactness, and we found that from the base of the coracoid to the loose fragment during extension of the forearm or contraction of the biceps measured two inches and three quarters, and measured from the anterior border of the clavicle to the fragment the distance was three inches. I wish now to be precise as to the direction of the displacement. Usually what is taught is that the displacement is in the direction of the resultant of three forces, represented by the three muscles attached to the process, viz. *downwards* and *inwards*. I take from the surgical part of Gray's 'Anatomy'[1] the following passage :—"Displacement is here produced by the combined actions of the pectoralis minor, short head of the biceps, and coraco-brachialis, the former muscle drawing the fragment inwards and the latter directly downwards." This may be taken as a scholastic view, probably supported by little surgical experience. I am always somewhat sceptical of purely anatomical surgery, and one need not go back to the anatomical romances of thirty years ago to learn how simple anatomical facts may be twisted to suit the most childish of theories. Let me now refer to Fergusson's experience,[2] that of a surgeon more practical than anatomical. "I have known an instance," he writes, "where the coracoid process of this bone was broken, and drawn *downwards* by the combined action of the pectoralis minor, coraco-brachialis, and biceps muscles; nothing could be done to keep the process in its natural position." The distinguished surgeon is here speaking of his clinical experience, and correctly describes a case similar to my own, in which wide separation of the fragments had taken place. He then introduces a diagram designed to show a fracture of

[1] Gray's 'Anatomy, Descriptive and Surgical,' 11th edit., p. 411.

[2] 'A System of Practical Surgery,' 5th edit., p. 239.

the coracoid process, but in describing this he soon falls into anatomical error. "The movable fragment," he continues, "would probably be more displaced than is here represented, especially if the pectoralis minor muscle should happen, as is shown in the drawing, to be attached lower than the second rib." The diagram correctly shows the usual origin of the pectoralis minor muscle from the third, fourth, and fifth ribs. What this distinguished surgeon intended to convey by suggesting it was usually attached higher than the second rib I am at a loss to conceive. His clinical observation, however, may be accepted without reserve.

Mode of Union.—If the comparison I have drawn between fracture of the coracoid process and those of the olecranon and patella be strictly correct, it should be shown now, or at some future time, that in a certain proportion of cases bony union may follow this injury. I am, however, unable to refer to any instance of the kind; but in those cases in which crepitus is easily obtained, and there is little displacement, it would seem that the lack of bony union must be referred to the treatment or lack of treatment, for its possibility if favoured by sufficient rest seems assured.

The following case is an instance of fibrous repair related by Mr. Shattock[1] concerning which there is no history:—"The specimen was obtained from an aged female subject. The fracture of the coracoid process is slightly oblique, being directed from before backwards, between the insertion of the pectoralis minor and the origin of the coraco-brachialis and inner head of the biceps; the coraco-clavicular ligament is attached solely to the entire base of the process. The fragments are united by a fibrous strap-like band above a third of an inch in length, and continuous with the adjacent portion of the coraco-acromial ligament. There was no injury of the shoulder-joint or of the clavicles. As in the cases cited by Flower and Hamilton, the detached fragment has undergone no material displacement, which indeed would probably be always true, since the coraco-clavicular ligament will detain the detached process in position should it be fractured through its base; or should the fracture pass, as in this specimen, in

[1] 'Path. Soc. Trans.,' vol. xxxii, p. 155.

front of the coraco-clavicular ligament, the detached portion will be as securely upheld by the coraco-acromial ligament."

The writer's anatomical argument against the possibility of wide separation of the fragments is disproved by the clinical experience obtained from my own case, Mr. Hulme's, and that quoted by Sir William Fergusson.

Specimens of fibrous union after fracture of the coracoid process are to be found in several of the hospital museums of the metropolis.

ON PRIMARY MALIGNANT DISEASE OF THE LIVER.

By W. HALE WHITE, M.D.

During the last few years I have twice made an autopsy on patients who have died from primary malignant disease of the liver, and I have had one patient suffering from this malady under my care. Dr. Perry made the autopsy in this instance. In all three cases there could not be the slightest doubt about the fact that the disease was primary in the liver, for absolutely no other organ could be found to be affected, although the whole body was most carefully examined; and in the case of the patient in whom the growth in the liver was melanotic nearly every mole on the body was subjected to microscopical examination, in order to make sure that it was not the seat of the primary disease.

The accidental circumstance of my having seen three examples of so rare a disease in a short time incited me to look up the clinical records of the cases we have had at Guy's Hospital; for while many instances have been shown at the Pathological Society during the last few years, they have been brought under the notice of members of the Society on account of their pathological interest, and not because of the clinical symptoms, which in the published accounts are either omitted or stated with great brevity. In selecting the cases which follow I have been careful only to choose those in which there could be no doubt that the disease was primary in the liver, as all the other organs in the body were examined, and found free from growth; the

only exception being that in some cases the lymphatic glands in the portal fissure or those on the upper surface of the liver were affected, clearly through secondary affection, and in one or two cases a minute nodule, which had obviously originated in the same way, was detected in the lower part of the lung. In consequence of this rigour in the selection of cases, some which are recorded in the post-mortem records as primary disease of the liver are not here included, because other organs were involved. It may have been true that in them the liver was the seat of the primary growth, still the implication of other organs naturally destroys the clinical value of the case, for it is difficult to know to what organ to assign some of the symptoms.

One of the most striking facts about these cases is that it is possible for a patient to die of malignant disease of the liver without the presence of any disease of the liver being suspected. Thus (Case 7) a woman aged thirty-nine was admitted into the hospital for what was regarded as the vomiting of pregnancy; there was no jaundice, and no one even thought of disease of the liver. Premature labour was induced, but the woman sank. The post-mortem examination revealed the fact that the liver was the seat of extensive malignant growth, but that all the other organs of the body were absolutely normal. A case almost parallel to this is recorded by Tivy [1] in which, had not the abdomen been examined and the liver found to be enlarged, it would have been impossible during life to suspect disease of this organ, yet the man quickly sank and died.

It would no doubt be desirable to group the cases according to the nature of the malignant growth, for in some it was cancerous, in others it was sarcomatous, and of the cancerous growths there were several varieties; but a perusal of the cases recorded in the Pathological Society's 'Transactions' will show how impossible this is, for often great doubt existed as to the nature of the growth, some thinking it was sarcomatous, others looking upon it as cancerous. Thus the specimen of melanotic malignant growth of the liver which I showed before the Pathological Society was regarded by all the members present as cancerous; but Professor Hamilton,

[1] 'Path. Soc. Trans.,' vol. xxxv, p. 237.

when he saw some slides of it which I asked him to look at, because in his work on pathology he says that cancers are never melanotic, said he was of opinion that the growth was sarcomatous. Then also in Dr. Coupland's[1] case, which is recorded in the Pathological Society's 'Transactions,' the view he took of the nature of the growth was different from that of the select committee to whom it was referred. In the face of such pathological uncertainty it is wiser for clinical purposes to include all the cases together under the title of primary malignant disease, although from carefully reading the recorded cases it seems to me certain that by far the larger number are cancerous.

The ten cases recorded at the end of this paper are the only instances which have occurred at Guy's Hospital during the twenty years 1870—1889, both inclusive. During that period there have been nearly ten thousand post-mortem examinations; that is to say that primary malignant disease of the liver is the cause of death in about ·1 per cent. of all the patients who die in a large general hospital. Probably the percentage is a little higher than this, because cases are not here included in which the growth was regarded as primary in the liver although present in other organs. During these twenty years there have been five such cases.

Many persons will no doubt be struck with the few cases of primary malignant disease of the liver we have had at Guy's Hospital, for during the five years 1885—1889, both inclusive, there have been autopsies performed upon sixty patients in whom the liver contained cancerous deposits secondary to cancer elsewhere. This would give 240 cases for the previous twenty years; and even if we allow that the cases in which there were growths elsewhere, but in which the primary seat was regarded as being in the liver, were genuine cases of primary malignant disease of the liver, that only gives fifteen cases of primary malignant disease of that organ, against 240 cases in which it was affected secondarily, or one case of primary for every seventeen of secondary growth; and if we take only the cases in which the growth was confined to the liver, we get one case of primary growth to twenty-four in which it was secondary.

[1] 'Path. Soc. Trans.,' vol. xxxi.

Frerichs[1] says, "As regards the relative frequency of primary and secondary cancer of the liver, and the proportion in which the several organs, in the case of secondary cancer, contribute to the development of cancerous deposit in the liver, my experience yields the following results:—Of thirty-one cases the cancer was confined to the liver and the adjoining lymphatic glands in five; in two cases it was accompanied by cancer of the lungs; in two by cancer of the lesser omentum and of the cœliac and cervical glands; and in one by cancer of the pericardium and pleura. In these cases the cancer in the other organs was so slightly developed that it was necessarily regarded as secondary to that in the liver. In addition to these ten cases of primary hepatic cancer, there were twenty-one others in which the disease of the liver was evidently of a secondary nature; of these there were ten in which it was consequent upon cancer of the stomach; one where it followed scirrhus of the pancreas; one where it accompanied scirrhus of the rectum; two where it accompanied cancer of the mammæ, and two where it accompanied cystic cancer of the ovaries; one where it was associated with medullary fungus of the retina; one with cancer of the brain; one with cancer of the retro-peritoneal glands; one with cancer of the anterior mediastinum; and one with epithelial cancer of the heel."

Thus of Frerichs's cases, the proportion of primary malignant disease of the liver without growth elsewhere to other cases of malignant disease of the liver was 5 to 26, or 1 to 5; and the proportion he gives when he has added cases observed by other authors is 22 out of 91. In the cases at Guy's the proportion was 1 to 24. If we take the cases regarded as primary in the liver but with growths elsewhere, the proportion of primary malignant disease of the liver to secondary disease is in Frerichs's cases 10 to 21, or 1 to 2; in the Guy's cases the proportion was 1 to 17.

It is to be specially noticed that Frerichs makes no allusion to primary disease of the bile-passages and gall-bladder, and this is probably the cause of the wide discrepancy between his figures and those from Guy's Hospital, for it is commonly allowed that one of the most frequent primary seats of cancer when the liver is implicated is the gall-bladder.

[1] 'A Clinical Treatise on Diseases of the Liver,' Syd. Soc. trans., vol. ii, p. 297.

The following analysis of the chief points of the ten cases we have had at Guy's Hospital brings out the leading features of the disease.

Age.—The fifth case, that of a boy aged twelve, had better be omitted from consideration here because, judging by his extreme youth and the exceptionally long duration of the disease, it is probable that his case is clinically at least an instance of some extremely rare affection of the liver, of which we know little. Of the remaining nine cases the eldest was seventy-one years old, and the youngest twenty-three. Five were more than fifty years old. Of seven cases recorded in the Pathological Society's 'Transactions' during the past twenty years in sufficient detail to be available, and not included in the ten cases from Guy's Hospital, the oldest patient was sixty-nine years old, the youngest thirty-three, and five were over fifty. We thus see that it is a disease of adult life, and generally of old age—a fact which is equally true of secondary cancer of the liver.

Sex.—Among the ten Guy's Hospital cases, five were men and five were women; and among the seven Pathological Society's cases two were women and five were men; so that among seventeen cases ten were men. This is interesting as confirming the fact that these were genuine cases of primary malignant disease of the liver, for had they been instances of primary disease of the gall-bladder and duct we should have expected the disease to be commoner in women, for gall-stones are much commoner in them than in men, and it is probable that cancer of the biliary passages set up by gall-stones is often a cause of cancer of the liver. Still it would be wise not to lay stress on so few cases as seventeen. In only one of the ten cases at Guy's Hospital were gall-stones found at the autopsy, and they were lying in the gall-bladder, which was healthy.

Family history.—In none of the clinical reports of these ten cases is there any mention that any of the patients' relatives had cancer. It is very difficult to obtain an accurate family history from hospital patients, but as there was no family history of cancer in any of the cases of primary malignant disease of the liver recorded by Murchison,[1] the point is worthy of further investigation.

[1] 'Diseases of the Liver.'

Duration.—Malignant disease of the liver is never a long-lasting malady, but when it is secondary to disease elsewhere the whole illness is considerably longer than when it is primary, for the first symptoms are due to the cancer in the organ primarily affected. When the liver is secondarily implicated it is often very difficult, and generally impossible, to say how long the cancer has been present in that organ before death. Still we all know from experience how long such cases will last, even after jaundice has developed, so that the jaundice has time to become darker and darker. Fagge says, "It is remarkable how long such cases linger, and even revive for a time when apparently at the point of death;"[1] and Frerichs says, "There are others where the disease is protracted over years."

Omitting Case 5, for the reasons already given when speaking of the age of patients, we find that after the first symptom appeared two patients lived four months, two lived three months, one lived two and a half months, two lived two months, and in one the duration could not be determined. That gives, roughly speaking, an average duration of twelve weeks. It is especially noteworthy that there are no wide limits of duration, so that it may be safely said that primary malignant disease of the liver is usually rapidly fatal, thus forming a striking contrast to those cases in which the organ is affected secondarily, and in which the patient often lingers for a long while. This conclusion is fully borne out by the cases recorded in the Pathological Society's 'Transactions,' for the durations in the cases in which it is mentioned are two, a half, three, and two months.

Pain.—This is nearly always present. In the Case 10, which was under my care, it was agonizing; sometimes it is very slight. The boy, who was only twelve years old, hardly complained of it at all. Dr. Burnet[2] records a case of primary melanotic sarcoma of the liver, and distinctly states that the patient felt no pain; and Dr. Tivy records the case of a woman who was suffering from this disease but had no pain.[3]

Tenderness.—The liver is usually mentioned as being tender; in Case 10 it was excessively so. Of Dr. Tivy's case it is

[1] Fagge, 'Principles and Practice of Medicine,' 2nd edit., vol. ii, p. 541.

[2] 'Path. Soc. Trans.,' vol. xxxvi, p. 252.

[3] Ibid., vol. xxxv, p. 227.

stated that the pressure must be very hard to produce any feeling of tenderness, and Dr. Coupland[1] records a case in which the organ was not tender. We may, however, conclude that the rule is for the liver to be both painful and tender.

Enlargement of liver.—The liver was always enlarged except in Case 3, in which it only weighed 36½ oz., this being one of the three examples of a contracting scirrhus of the liver resembling cirrhosis. I found that among fifteen cases which are available, in the Guy's Hospital and the Pathological Society's cases, for calculating the weight, the heaviest was 267 oz., the next 200 oz., and the least 36½ oz. The one of 200 oz. was the exceptional case in the boy aged twelve, and the one weighing 267 oz. is recorded as a case of sarcoma. If we exclude these two the average is 116 oz., or if we only exclude the boy the average is 127 oz. A very usual weight is from 120 to 130 oz. The surface is hard and nodulated.

Jaundice.—This symptom is thus referred to:—Case 1. Slight jaundice. Case 2. No mention. Case 3. Conjunctivæ yellow. Case 4. No decided jaundice till two days before death. Case 5. Slight jaundice a month before death; it increased. Case 6. No jaundice. Case 7. No jaundice. Case 8. Much jaundice. Case 9. No jaundice. Case 10. Became jaundiced shortly before death.

Thus we see that out of ten cases, in four there was no jaundice, in two others it did not appear till just before death; in three cases it was slight, and in one case it was considerable. Out of seven Pathological Society's cases it is only mentioned as being present in four cases, and in one of these it was slight. We may thus conclude that in primary malignant disease of the liver, jaundice may be absent all through the illness; if present it is usually slight, and comes on late in the illness. We never meet with the long-lasting dark jaundice so common when the liver is affected with secondary cancer. The explanation of this may lie in the fact that as the disease is rapidly fatal, there is not time for jaundice to supervene. It cannot be due to the circumstance that the portal glands are rarely enlarged in primary carcinoma of the liver, for in the only case in which the jaundice was deep it is expressly stated that the portal glands were normal. Probably they

[1] Ibid., vol. xxxi, p. 180.

are not more frequently enlarged because the patient dies before there is time for infection to take place.

Ascites.—In six of the ten Guy's Hospital cases there was ascites, and in most of these it was sufficient to be detected during life; possibly in the remaining four there was a little serous effusion into the peritoneal cavity, but it was not striking. Among the Pathological Society's cases ascites is only definitely mentioned as being present in two cases out of seven during life, and in one other a little fluid was found at death. Probably we shall be near the mark if we say that in about a third of the cases there is definite ascites capable of detection during life, and in rather over a half serous effusion can be found after death. The growth often grew into and caused thrombosis of the branches of the portal vein, and this may in some cases explain the ascites. Often, no doubt, in ordinary cancer of the liver, cancerous peritonitis is largely answerable for the ascites; but that cannot be so in these cases, for in none of them was the peritoneum affected, except the small portion which covers the liver.

Temperature.—In Case 2 the temperature ranged between 101° F. and 104° F. Towards the end of Case 5 the temperature was high, and before death it reached 105·2° F. In Case 9 it occasionally reached 101° F.; and in Case 10, in spite of extreme collapse, it was 99° F. The temperature is mentioned as being raised in one of the Pathological Society's cases. Fagge mentions that there is occasionally a rise of temperature in cancer of the liver, and Dr. Carrington[1] has observed pyrexia in eighteen out of forty-four cases of cirrhosis. Murchison[2] mentions two cases in which there was pyrexia, and it is noteworthy that in both of them the cancer was primary in the liver. Taking cancer of the liver as a whole, Murchison looks upon a rise of temperature as decidedly rare. The cases here mentioned rather indicate that it is commoner when the disease is primary in the liver than when it is secondary. If this should turn out to be so it may be of some value as a means of diagnosis.

Other symptoms.—As might be expected, the patients are often wasted, sometimes they vomit; often there is constipa-

[1] 'Guy's Hosp. Rep.,' vol. xlii, 1883.

[2] Op. cit.

tion, but the stools are never mentioned as being pale. If the jaundice be sufficient a little bile may be detected in the urine, which in two out of the ten cases contained albumen.

From these cases we may, I think, gather that there are three varieties of primary malignant disease of the liver.

Firstly, there is the extremely rare and probably unique case of the child (Case 5) aged twelve in whom the disease lasted eighteen months, and the temperature reached 105·2° F. This case is alluded to by Fagge, and has been placed on record by Dr. Pye-Smith.[1]

Secondly, there are those cases of scirrhus which resemble a cirrhotic liver. Cases 1, 2, and 3 are instances of this variety, and they will be found recorded by Fagge in the Pathological Society's 'Transactions.'[2]

It will be noticed that all these three cases were in men, and that the duration of the disease from the first symptom was two and a half, three, and two months. They all complained of pain. In the two in which the liver could be felt it was tender, nodular, and hard. In all three there was ascites discoverable during life. Two were jaundiced, they were all wasted. Their ages were respectively seventy-one, forty-six, and fifty-two years; none of them were drinkers. At death the livers weighed 180 oz., 62 oz., and 36½ oz. We have had other cases at Guy's Hospital in which the prevailing opinion has been that they were instances of primary cancer rather than cirrhosis, but as there has been some doubt about the matter I have not included them here.

Thirdly, we have the remaining cases, which constitute the most common form of primary malignant disease of the liver: except for the accidental resemblance to a cirrhotic liver there is no valid reason for separating the second group from this; it is only because it is so important to bear in mind the fallacy that I have done so.

Taking, therefore, all the cases of primary malignant disease of the liver together, except the case of the child, we find that for the most part it is difficult to distinguish them from in-

[1] 'Path. Soc. Trans.,' vol. xxxi, p. 125.

[2] Vol. xxviii, p. 137.

stances in which that organ is affected secondarily. In both varieties the disease occurs in elderly men and women, and leads to wasting. The liver is usually painful, tender, enlarged, hard, and nodular. Jaundice, ascites, sickness, and constipation are often present.

There is, however, one important point which may help in a diagnosis, and that is the uniformly rapid course of the primary cases. Out of seventeen cases the longest duration from the first symptom was four months, the average duration is twelve weeks, and it may be as short as eight weeks or less. This probably accounts for the fact that the jaundice is often absent, and that if present it is usually slight, and that the stools are never pale; and in connection with this it is interesting to notice that the lymphatic glands in the portal fissure are not much enlarged. These patients never present the characteristic dark jaundice of long-standing secondary malignant disease of the liver. Consequently this collection of cases may help us in the future to form a correct diagnosis, for we may probably affirm of any case that has lasted more than four months that the disease of the liver is secondary. Perhaps, too, further experience will teach us to predict primary malignant disease of the liver if the temperature is considerably elevated. In many cases of secondary cancer of the liver, in which the jaundice is very deep, the patients die from cerebral symptoms which are grouped together under the name of cholæmia. As the jaundice in cases of primary malignant disease of the liver is often absent, or at least but slight, it is probable that when the disease is primary the patients do not die from reabsorption of bile.

Case 1.—Fred. McC—, æt. 71, admitted into Clinical Ward under Dr. Habershon, October 12th, 1874. Is a perfumer. Father died at ninety, mother at seventy. No history of malignant disease.

Previous history.—Always had good health till three years ago, when he vomited some blood. Quite well from then till a month ago, when he first felt pain in stomach, and noticed a lump in the hepatic region. No drink nor syphilis.

On admission.—Thin, slightly jaundiced. Liver painful, tender and enlarged to anterior superior iliac spine, nodular,

especially lower part. The tumour increased in size, the pain became worse, the jaundice increased, and the patient died November 24th. Shortly before death a fluid thrill could be obtained in the abdomen. No mention of bile in the urine or pale stools. He had diarrhœa.

Autopsy.—A quantity of blood-stained fluid in the abdomen, which on the right side in the region of the liver contained blood-clot. The liver was adherent to the diaphragm. It weighed 180 oz., and was nodulated with small nodules. These were scattered through the organ, and were obviously cancerous. Some were cheesy, so that they would shell out; some of the larger masses had hæmorrhage in them, and the blood in the abdomen obviously came from the liver. Aorta atheromatous.

Case 2.—John W. E—, æt. 46, admitted into Philip Ward under Dr. Fagge, 26th October, 1875. Is a rope manufacturer. Father died of rheumatic fever, mother of old age. No history of malignant disease.

Previous history.—Always had good health except for a bad attack of gonorrhœa. Nine weeks ago first fell sick. Seven weeks ago first felt pain in the stomach. Two weeks ago noticed that his abdomen began to swell. Has drunk freely.

On admission.—Six feet high, ten stone in weight. Temp. 102·4°. Tongue furred. Abdomen distended and resonant all over; no evidence of fluid. Liver and spleen not distinguishable.

November 7th.—Temperature has ranged between 101° in the morning and 104° in the evening. The abdomen is still distended, but there is distinct fluctuation, and the liver can be felt as a tender, hard, nodulated mass. The abdominal veins are much distended. The temperature continued to range as just mentioned till the patient's death on November 10th. No mention of jaundice. Bowels once mentioned as being constipated.

Autopsy.—The abdomen contained a large quantity of serous fluid. The liver weighed 62 oz. It was adherent to the abdominal wall. The part most diseased was the anterior lobe, which was the seat of a diffuse carcinoma. It had grown into

the portal vein, and given rise to thrombosis. In many parts of the liver were soft rounded masses of cancer, each with a fibrous covering. The liver tissue itself was tough. The gall-bladder was healthy. There was no trace of carcinoma anywhere in the body.

Case 3.—John B—, æt. 52, admitted into Stephen Ward under Dr. Pavy, September 22nd, 1876. Is a gardener. Father died of fever, mother of old age. No family history of malignant disease.

Previous history.—No previous illnesses. A month ago first noticed a stiffness in the abdomen, and three weeks ago he noticed it was enlarging. Soon after he began to complain of pain.

On admission.—Is emaciated. Conjunctivæ yellow. Abdomen very distended by fluid, veins not marked. Feet and scrotum swollen. Liver and spleen not to be made out. Urine normal. Temperature normal.

October 14th.—There has been much constipation, but no pain. Breathing has been very difficult. Fourteen pints sixteen ounces of straw-coloured fluid were withdrawn. Even after this the liver could not be felt. Fluid re-collected, and patient died on the 18th.

Autopsy.—The liver was contracted and nodulated, weighing 36½ oz. It was very hard, and looked just like a cirrhotic liver, except that the nodules, some of which were as large as currants, were more definite in outline than is usually the case in cirrhosis. On section the organ was found to be wholly made up of similar rounded masses of what appeared to be altered hepatic tissue, most of them brown in colour, but some whitish yellow. Here and there one had a dry cheesy look, and on pressure its substance could be squeezed out, leaving an ill-defined capsule, which seemed to consist of the fibrous material, forming septa which traversed the organ in all directions. These spots certainly suggested carcinomatous growth, but none of them were as big as peas. The portal vein was thrombosed. Microscopically the case was clearly one of cancer with large polymorphic cells.

Case 4.—Jane H—, æt. 56, admitted into Addison Ward under Dr. Pavy, February 26th, 1877. No occupation. She

is married. Mother died at sixty, father from an accident. No history of malignant disease.

Previous history.—No previous illnesses of any importance. Three and a half months ago began to suffer from pain in the epigastrium, constipation, and sickness. These symptoms continued till admission.

On admission.—Whether the sclerotics are jaundiced is doubtful. Abdomen distended, no fluctuation; liver can be felt below the umbilicus; it is tender; no venous distension. Urine albuminous. Constipation. Abdominal pain. Temp. 97·6°. After a time she became a little better, and the albuminuria disappeared. By March 10th she was jaundiced. On 12th she died; she suffered much from pain and vomiting.

Autopsy.—The omentum was adherent to the liver, but was not infiltrated with growth. The stomach contained two superficial ulcers, certainly not malignant. The liver was enormous, weighing at least 120 oz. It was studded throughout by large and small rounded nodules of cancer; many of them had coalesced to form larger masses. The liver tissue was soft and deeply jaundiced. The growth was extremely fibroid, and formed alveoli in which were embedded epithelioid cells. The gall-bladder contained a few gall-stones, but was otherwise healthy. There was no trace of any growth in any other part of the body.

Case 5.—William T—,[1] æt. 12, admitted under Dr. Pye-Smith, November, 1878. Three months ago he noticed a swelling in the region of the liver which sometimes gave him pain. The liver was firm, smooth, elastic, and the edge was three to four inches below the ribs. There was no ascites nor jaundice. A puncture only brought away a little bloody fluid. In September, 1879, he was readmitted; his condition was unchanged; he complained occasionally of pain, giddiness, or vomiting. He was admitted for the third time in January, 1880. He was thinner, the liver was much larger, but he had little or no pain, and he was free from vomiting. There was a slight tint of jaundice. On the evening of February 13th he vomited, and his temperature was 100·2° F. Next morning it was 104·6° and there was more jaundice and slight albumin-

[1] Recorded by Dr. Pye-Smith, 'Path. Soc. Trans.,' vol. xxxi, p. 125.

uria. On 15th the temperature was 103°, on 16th it was 105·2°. He died on 16th.

Autopsy.—The abdomen contained some straw-coloured fluid. The liver weighed 200 oz. The right lobe was greatly enlarged, and full of more or less circumscribed masses of soft yellow material, which had grown into the portal and hepatic veins. These new growths varied from microscopic dimensions to huge infiltrating masses. One large tumour had completely blocked the portal trunk and compressed the common bile-duct. Hæmorrhage had occurred in a few, and all were more or less degenerated. The glands in the portal fissure were normal. With the exception of a small nodule at the base of the right lung there was absolutely no new growth anywhere in the body. The microscope showed it to be a carcinoma.

Case 6.—Isabella K—, æt. 60, admitted into Addison Ward under Dr. Pavy, December 17th, 1879. Is married, has no children. Family history unimportant.

Previous history.—Has had rheumatic fever; six months ago suffered from shortness of breath and cough; got better of this. A month ago vomiting began, and three weeks ago the swelling of the abdomen commenced, together with pain in the hepatic region.

On admission.—No jaundice. Liver painful, enlarged, and nodular. No albuminuria. No evidence of heart disease save a slight diastolic murmur heard down the sternum. Soon after admission fluid began to collect in the abdomen, and it had to be drawn off. Vomiting, constipation, and pain during nearly all her illness. She died March 6th, 1880.

Autopsy.—The liver weighed 91 oz. The liver tissue in the right lobe was almost converted into a mass of very dense grey cancer; in the left lobe it was scattered about in circular nodules, some of which had undergone caseous change. The growth was composed of small angular cells, each with a large central nucleus. The gall-bladder was healthy.

The pleuræ each had a small thin patch of cancer growth in them at the posterior part. The mediastinal glands were slightly enlarged and distinctly cancerous. The rest of the body was healthy. Dr. Goodhart regarded the case as one of primary carcinoma of the liver.

Case 7.—Martha L—,[1] æt. 39, was admitted into Guy's Hospital under Dr. Galabin, November 12th, 1884, for the vomiting of pregnancy at the third month; soon afterwards she went out, but was readmitted in the sixth month of pregnancy with albuminuria, vomiting, dyspnœa, œdema of the legs, and a systolic bruit. Premature labour was induced, but the patient sank and died soon after. There was no jaundice nor any other symptom which suggested disease of the liver and could not be explained by the fact of her being pregnant.

Autopsy.—The liver weighed 126 oz.; it was very pale, fatty, with rounded edges, and had several malignant lumps scattered in it. These were soft, hæmorrhagic, and light yellow in colour; a single lymphatic gland in the transverse fissure of the liver was slightly affected. The mitral valve was perhaps slightly thick, but hardly abnormal. With the above exceptions the whole body was absolutely normal.

Case 8.—Sarah G—, æt. 23, was admitted into Mary Ward under Dr. Pye-Smith, August 23rd, 1884, with hard lumps in the liver and much jaundice. No primary source could be detected in the breast, uterus, &c. She gradually sank, and died September 22nd, 1884.

Autopsy.—All the organs of the body were deeply jaundiced. The liver weighed 125 oz.; it was irregularly nodulated by large whitish masses of carcinoma, hard in parts, but in others so soft that it consisted only of a dark reddish-brown muddy mass. Some of the nodules were full of recent blood. They were very variable in size. The largest was as large as a small cocoa-nut. No organs were adherent to the liver. The gall-bladder was quite healthy. The portals glands were not enlarged. Every part of the body including the eyeballs was examined, but no primary source could be found.

Case 9.—George C—,[2] æt. 66, was admitted into Guy's Hospital under Dr. Pavy, October 2nd, 1885, for hepatic enlargement and wasting. There was no family history of malignant disease. The right eye had been excised a year before for glaucoma. Mr. Mackinlay, who removed it, told

[1] 'Path. Soc. Trans.,' vol. xxxvi, p. 251.

[2] 'Path. Soc. Trans.,' vol. xxxvii, p. 272.

me there was no suspicion of malignant disease. Two months before admission he began to complain of pain in the hepatic region, and then he noticed a swelling there which was tender. At no time during his illness was thery any jaundice, nor had he albuminuria or vomiting. There was slight constipation. The temperature occasionally reached 101° F.

Autopsy.—The liver weighed 122½ oz. It was much enlarged, especially the left lobe, and the left-hand two thirds of the right. The rest of the liver was normal except that there were scattered through it the black hard nodules of new growth, varying in size from a mere speck to three quarters of an inch in diameter. In many places they were umbilicated. There was one large and one small white mass without any pigment. The rest of the liver was slightly rough and adherent to the diaphragm; it was almost black, slightly tinted with grey and green. The liver substance was nearly entirely replaced by new growth, which was hard and had absolutely black lines running through it, and giving the whole a foliaceous appearance. It had slightly invaded a branch of the portal vein. Some glands on the upper surface of the liver were deeply pigmented. The glands in the portal fissure were slightly enlarged and of a dark colour.

Excepting for the atrophy of the contents of the right orbit the whole of the body, which was examined in every part, was absolutely normal. Microscopically the growth appeared to me, and to everyone to whom I showed it except Professor Hamilton of Aberdeen, to be a melanotic carcinoma. Professor Hamilton considered it to be a melanotic sarcoma. Several moles from the skin were examined microscopically, but they did not show any growth.

Case 10.—Fanny L. H—, æt. 29, admitted under Dr. Hale White, October 14th, 1889. No history of malignant disease; has always been weakly. Eleven weeks ago noticed a lump in the stomach. On admission is in great pain; no jaundice; much wasting. Liver big and hard, tender, and nodular. Temp. 99°. Four pints were drawn off from abdomen soon after admission, but she was so collapsed that paracentesis had to be stopped. No vomiting. No albumen, but much bile in urine; became jaundiced shortly before death.

Autopsy.—There was no general peritonitis; the adhesions of the liver were unimportant, omentum free. In the peritoneal cavity was about a pint of bile-stained fluid, non-purulent; weight of liver 8½ lbs. Enlargement uniform, surface irregular; in one place distinct umbilication. On section circumscribed cancerous deposits. The liver and growth were stained with bile. The wall of the gall-bladder was implicated. There was some hæmorrhage into the growth, some of which grew into the lumen of the portal vein. There was no trace of growth anywhere else in the body. The microscope showed the ovaries and mammary glands to be normal. Except for the cancer in it the liver was normal. No lymphatic glands affected in portal fissure or elsewhere.

THE PATHOLOGY

OF

ACUTE INFECTIVE PERIOSTITIS AND ACUTE NECROSIS OF GROWING BONE.

BEING THE REPORT OF AN INVESTIGATION CARRIED ON IN THE WORKROOMS OF THE ROYAL COLLEGE OF SURGEONS OF ENGLAND AND THE PATHOLOGICAL INSTITUTE AT LEIPZIG.

By A. H. TUBBY, M.S.

The object with which this work was undertaken was the determination of the more exact etiology and pathology of that most grave disease of osseous tissue generally designated in surgical nosology acute infective periostitis, or acute necrosis. This disease is characterised by its sudden onset in children and adolescents, often with typhoid-like symptoms, the rapid course of the disease, the early formation of pus, the frequent death of a large part of the bone, and in some cases by the complication of pyæmia. To carry out the inquiry some experiments were made in the Pathological Institute at Leipzig, and the results examined microscopically through the generosity of the Council in the workrooms of the Royal College of Surgeons.

To those gentlemen who have allowed the writer to search for notes of their cases, and to make use of them, and for permission to examine specimens in museums, he tenders his grateful thanks.

Acute inflammation of bone is universally accepted to be of most common occurrence during the periods of childhood and youth, and the following remarks on the structure of bone are confined to this time of life. It is found that during these periods the shaft and epiphyses are not continuous, but separating them is a layer of proliferating tissue, the epiphysial line.

Structure of Growing Bone.

The various parts needing notice are—(1) the periosteum, (2) the ligaments, (3) the epiphysial line, (4) the synovial membrane together with the articular cartilages.

The periosteum is composed of two layers, superficial and deep. The former consists of fibrous tissue, supporting the larger blood-vessels of the membrane; while in the latter the fibrous tissue is more loosely arranged, and in its meshes are a number of large granular cells (osteoblasts), together with the smaller vessels, nerves, and lymphatics of the periosteum.

The superficial layer if traced downwards towards the joint becomes directly continuous with the capsular and other external ligaments of the joint, while the deeper is seen at the extremity of the diaphysis to divide, the larger portion passing inwards and its cells undergoing transition; they become continuous with the cartilage-cells of the epiphysial line, so that the ends of the diaphysis are enclosed in a cap of young growing tissue formed externally of the deeper layer of the periosteum and internally of the epiphysial line. The other portion of the deeper layer passes toward the joint-cavity over the epiphysis, and is there continuous with its permanently unossified part, the articular cartilage. Kölliker[1] has further demonstrated that during fœtal life the epiphysial cartilage is prolonged into the tissues which are developing into synovial membrane; and Heuter[2] has also shown that the synovial membrane is structurally continuous with the articular cartilage, the cartilage-cells of the latter gradually assuming the appearance of connective-tissue corpuscles at the point of contact of the two structures. Of this fact we have pathological

[1] 'Bemerk. und Ossification,' Leipzig, 1873.

[2] Quoted by Macnamara, 'Diseases of Bones and Joints,' p. 12.

proof in the formation of osteophytes in the fringes of the synovial membrane in osteoarthritis.

With regard to the minute structure of the growing line a fuller description is called for. It is seen to be composed of the following four zones or layers.

The first is the proliferation line (Proliferationschicht) of Strelzoff,[1] and comprises the distal part of the cartilage. It is composed of hyaline material with small cells embedded, which are distally spindle-shaped, but towards the shaft become wedge- and half-moon-shaped. (This is Kassowitz's[2] line of general cell proliferation.) Still nearer the diaphysis are seen columns arranged parallel to the long axis of the bone (nearly in the same position as Kassowitz's "layer of partial cell proliferation"). The second layer, described by Strelzoff as the "layer of hypertrophy," comprises many large rounded cells with big nuclei which have burst through the cartilage-capsules and are embedded in only a small amount of hyaline substance. In the third layer, the "calcification layer," there is a deposition of calcareous material in the ground-substance, and the cartilage-cells have undergone thickening and wrinkling before their final disappearance. The fourth or granulation zone of Strelzoff, Kassowitz's bone-forming layer, is placed between the young bone and the calcified cartilage.

It consists of two or three rows of cavities which originate from the destruction of the cartilage-capsules, and are occupied by medullary substance. These cavities are quite filled by numerous soft medullary cells (osteoblasts) of very delicate structure, and here the blood-vessels terminate in capillary loops, which are often somewhat dilated. The importance of this last layer of embryonic, and therefore unstable material, should not be underrated in the consideration of the origin of bone diseases. Prone to great normal activity, it is also subject very readily to any abnormal influence, and it is "but reasonable to expect that it will be the first to show pathological changes, and that the changes will be most marked here."[3]

[1] 'Ueber die Histogenese des Knochen,' Leipzig, 1873.

[2] 'Die Normale Ossific.,' Wien, 1881.

[3] Macnamara, op. sup. cit.

We know that in traumatic separation of the epiphyses the line of rupture passes directly through this zone, leaving the cartilage attached to the epiphysis. This zone will hereafter be styled for brevity the "juxta-epiphysial junction."

Bearing upon the question of its proneness to respond to abnormal stimuli, the appearances seen in syphilitic epiphysitis may be cited. Not only is the multiplication of cells so great as to obscure the normal outline of this region, but even the neighbouring cartilages is invaded by the new young cells, and the proper cartilage-cells have disappeared, to be replaced by these inflammatory cells.

Literature.

Under the heading of "Osteomyelitis" Mr. Jones, of Manchester, in his recently published work on 'Diseases of the Bones,' gives an exhaustive résumé of the literature of the subject. Cruveilhier[1] in 1816 was the first to draw attention to the presence of pus in the medullary cavity, especially after amputation; but to Stanley[2] the credit is due of describing osteomyelitis more precisely. Chassaignac[3] in 1853 gave a classical description of osteomyelitis arising in young subjects. In 1855 Klose[4] of Breslau described thirteen cases of acute Epiphysentrennung (separation of epiphyses), or meningophlebitis, a description characterised by Jones as one-sided. Gosselin[5] stated that the disease originates in that part of the bone adjoining the cartilage between the epiphysis and diaphysis, and designated it "Epiphysary ostitis." Frank's[6] description resembles Chassaignac's, and he calls the disease "Inflammatory epiphysary ostitis." Demme[7] draws a distinction between osteomyelitis accompanied or not by phlebitis,

[1] 'Pathol. Anat.,' Paris, 1816.

[2] Stanley, 'Diseases of Bones,' pp. 18 and 34, 1849 edition.

[3] 'Trans. Acad. Sci.,' 1853.

[4] 'Vierteljahrschrift für die praktische Heilkunde,' Jahrgang 15, Band i.

[5] "Sur les Ostéites epiphysaires des Adolescents," 'Archiv. Gén. de Méd.,' Nov., 1858, p. 513.

[6] 'Ueber Entzündliche Epiphysenlösung,' Inaug. Diss., Giess., 1861.

[7] 'Archiv für klin. Chirurg.,' 1862.

and states that the former is the more malignant, an observation of much value now that the rôle of pyæmic thrombosis and infarctus has been defined. Lannelongue's work in 1879 urged the necessity of early operative treatment. Recently Ollier[1] has applied the term "acute specific juxta-epiphysitis" or "acute diaphysitis." Amongst English surgeons Mr. Holmes's[2] name is closely associated with the subject. Writing in the 'System of Surgery,' he says, "How far it is possible to distinguish acute periostitis from acute osteomyelitis is a question on which surgeons differ widely. Many French and German surgeons hardly admit the existence of the disease which has been known as diffuse periostitis apart from osteomyelitis, of which they consider it a complication. Some assert that the whole thickness of the shaft of the bone is never affected unless the medullary canal is involved." "But," he adds, "I am confident that cases of acute periostitis do occur in which the whole thickness of the shaft (and sometimes the entire shaft) perishes, without any implication of the medullary membrane, although I allow that in most cases of diffuse periostitis implicating the whole thickness of the bone, the medullary tissue is more or less involved." (It appears difficult to understand how it is possible for the entire shaft to die, and yet no implication of the medulla to occur, when we remember that the medulla internally plays the same part as the periosteum externally, viz. that of a supporting membrane for vessels destined to nourish the inner layers of compact bone near to it. In the last sentence it is admitted that in most cases the medulla is involved.)

Mr. Bryant[3] says that acute inflammation of bone is periosteal or endosteal. If periosteal alone, an outer shell of bone only may die; if endosteal, the periosteum is sure to be implicated, and the greater part of the bone if not the whole shaft will be necrosed.

Mr. Erichsen[4] remarks, "There is no doubt that in fatal cases it is very common to find the medulla inflamed and often suppurating, but this is by no means constant and often

[1] 'Archiv. de Médécin,' 1875.

[2] 'System of Surgery,' 3rd edit., vol. ii, p. 286.

[3] 'Surgery,' 1881, vol. i, p. 881.

[4] 'Science and Art of Surgery,' 8th edit., vol. ii, p. 280.

secondary; in many cases, *e. g.* in a case at University College Hospital, a section of the bone showed the medulla to be inflamed on one side only, in contact with the part of the compact tissue which was denuded of its periosteum. . . . . The inflammation was evidently of a more recent date than that of the periosteum. The disease is supposed to be due to a virus entering from without and finding the nidus for its development in the vascular growing tissue beneath the periosteum, or in the layer of cartilage between the shaft and the epiphysis, the determining cause of the local outbreak being in most cases a slight injury with extravasation of the blood."

Ashby and Wright,[1] in a recent work on 'Diseases of Children,' state that as the accounts of different writers on the subject of acute bone inflammation are somewhat conflicting and have given rise to some confusion, so the accompanying tabular statement of how the different lesions may arise will perhaps be of service to those less familiar with bone diseases.

Acute inflammation of bone may begin as—

A. Periostitis.
   1. Subperiosteal.
   2. Supra-periosteal or parosteal.

B. Osteomyelitis.
   1. Epiphysitis, *i. e.* disease beginning in the cancellous tissue of the epiphysis.
   2. Inflammation beginning in the medulla of the shaft (diaphysitis).
   3. Inflammation beginning in the epiphysial line, often called epiphysitis also.

They add, "Inflammation beginning in any of these ways may give rise to the other forms of the lesion; *e. g.* subperiosteal abscess may spread along the epiphysial line, and, causing suppuration of the medulla of the shaft or inflammation of the medulla, may spread outwards and cause periostitis." They believe "acute periostitis is not really acute osteomyelitis, since, if it were so, complete recovery without extensive necrosis would not be nearly so common as it is in these cases."

The truth of this last remark depends on the date and nature of the treatment.

[1] 'Diseases of Children,' 1889, p. 497.

Mr. MacNamara is the most vigorous English exponent of the Continental view that acute infective periostitis, accompanied by total destruction of the shaft, is due to inflammation primarily of the soft tissue at the extremity of the shaft, *i. e.* it commences as a juxta-epiphysitis. He read at the Royal Medical and Chirurgical Society a paper on the subject, and brought forward specimens which formed a striking confirmation of his opinion. In the debate he was stoutly opposed by Mr. Holmes.

Mr. Jones[1] says that "acute inflammation of bone may commence either in the medulla or the periosteum, very rarely in the osseous tissue proper. Experience seems to show that the medulla is a very frequent starting-point of the traumatic inflammatory disorders of bone." Again, on page 129 it is stated that "the ostitis which produces necrosis is often acute, and follows acute osteomyelitis or acute periosteal abscess."

What are the conclusions to be drawn from a *résumé?* On the one hand, there are Continental authorities consistently affirming that acute periosteal abscess is a secondary result or a complication of a primary inflammation in the medulla or in the juxta-epiphysial region : on the other hand, our home authorities bid us look to the periosteum as the starting-point of the disease; yet others speak with reservation, and are of opinion that acute necrosis may commence in either tissue.

Experimentally, Troja, in 1775, by destroying the marrow of the diaphysis, observed that the necrosis which followed corresponded in extent to the amount of medulla destroyed, the periosteum also being detached to an equal extent.

Ollier states that under favorable circumstances removal of the periosteum alone does not give rise to necrosis, but that a reproduction of periosteum from surrounding tissue occasionally follows; and in animals even the simultaneous removal of both periosteum and marrow is not always followed by necrosis. It is not stated whether the juxta-epiphysial junction was left intact; but if it were so, the shaft can still obtain vascular supply from the numerous vessels passing between epiphysis and diaphysis. And the writer thinks that if we are to explain satisfactorily the mode of onset of the

[1] Op. cit., p. 31.

disease, the concurrence of periostitis and endostitis, the early and complete separation of the shaft with necrosis of its whole thickness, some region of bone other than the periosteum or medulla must be the site of earliest inflammation. This region is believed to be that vascular layer of young growing bone, the juxta-epiphysial junction. Inflammation commencing here spreads upwards to the medulla and outwards to the periosteum, assisted by the peculiar disposition of this membrane at the epiphysial line.

It is impossible to believe that in all so-called cases of acute periostitis the direction of the violence is always such as to fall directly on to the periosteum, or so great as to involve its separation from the bone. More often the violence is indirect, and yet while of such a nature as to be insufficient to cause grosser lesions, such as fractures, dislocations, and separation of the epiphyses, it occasions some wrenching or twisting in the length of bone which obviously can affect but slightly the thick and tough periosteum, but exerts its full effect upon the weakest and most vascular part of the growing bone—the juxta-epiphysial junction. The more frequent occurrence of necrosis in the lower limbs points to this, since in childhood and youth the bones are constantly exposed not only to direct violence, but still more to indirect violence in leaping and running. Tired children, says Gosselin, are very liable to osteomyelitis. Which structure is more likely to suffer injury—the periosteum, or the epiphysial line with the parts in its neighbourhood which are continually acting as cushions to ward off shocks to the vertebral column from the impact of the feet on the ground in walking? The writer then would advance the following statements for consideration:

1. The disease variously known under many synonyms, but for convenience here called acute necrosis, and characterised by the definite train of symptoms mentioned on p. 77, is due to the irritation and suppuration occasioned by the entrance and development of micro-organisms, the *Staphylococci pyogenes aurei;* and further, that it is this specific acute disease and not merely simply traumatic periostitis which gives rise to the death of the shaft.

2. The *earliest* symptoms of the disease appear in the *juxta-*

epiphysial regions, and spread thence inwards to the medulla and outwards to the periosteum.

3. That in cases of total death of the diaphysis the destructive process is most marked at the extremities, the bone here being found eroded and gangrenous; and further, that there is such a loss of tissue that the dead shaft no longer fits accurately into the epiphysial and periosteal cap.

4. If the origin of the disease as a juxta-epiphysitis be conceded, then a more thorough and radical method of treatment is necessary; and operators must not be merely content with incising the periosteum and letting out the pus, but should attack the disease at its very root, and, by tunnelling the juxta-epiphysial junction in addition, allow a sufficient exit for the products of the inflamed medulla.

5. A few remarks will be made upon the channels of ingress of the micrococci into the blood other than the respiratory and digestive tracts, and the bearing of such other modes of ingress upon the disease in question.

First.—Commencing with statement No. 1, acute necrosis is due to the irritation caused by the entrance of the *Staphylococcus pyogenes aureus.* It is now well established that osteomyelitis is accompanied by "purulent infection." Klebs[1] described in 1873 the spores of an organism found in a case of osteomyelitis which he was able to cultivate, and his investigations were followed by those of Lücke[2] in the next year. He found in the purulent discharge from bone infection a micrococcus corresponding in all respects to that found in the secondary centres.

Pasteur a few years after discovered an identical organism in a case of osteomyelitis under Lannelongue's care, and further noted its resemblance to a similar one found in boils.

The most conclusive experiments are those of Becker,[3] who not only cultivated the *Staphylococcus pyogenes aureus,* but found that intravenous injections of the micrococci produced a fatal result with osseous suppuration whenever there existed fracture or contusion of bone. Rosenbach extended these results, and concluded that another microbe, the *Staphy-*

[1] 'Virchow's Archiv,' 1875, Bd. xlv, p. 352.

[2] 'Archiv für exp. Path. und Pharmacie,' Band v, s. 31.

[3] 'Berlin klin. Wochenschrift,' Dec., 1883.

lococcus pyogenes albus, was occasionally found. Dr. Krause,[1] of Halle, further confirmed these investigations. He noticed that osteomyelitis so produced had a peculiar tendency to attack the joints and to give rise to suppuration in muscles. A good illustration of this observation is seen in one of the writer's experiments, viz. No. 2.

Rodet[2] found a microbe in the pus of osteomyelitis, and Krause and Pasteur found a similar one in discharges from boils, while Garre detected them not only in boils, but in phlegmon and whitlow. Rosenbach looks upon the microbe in osteomyelitis as identical with that found in ordinary pus, Garre[3] with the microbe of osteomyelitis induced boils, and Rodet induced osteomyelitis with the microbe of boils.

"Kraske[3] concludes that acute osteomyelitis in man may be produced by the *Staphylococcus pyogenes aureus* alone, but that if the *Staphylococcus pyogenes albus* and *Streptococcus pyogenes aureus* and bacilli are found it indicates a peculiarly severe type."

(For much of the three foregoing paragraphs the writer is indebted to a careful *résumé* of the subject by Mr. Jones, of Manchester, in his work on 'Diseases of Bone.') With reference to the presence of the *Staphylococcus pyogenes aureus* in pus from cases of acute infective periostitis[4] the writer has frequently ascertained its existence, and he has been able to identify it satisfactorily with the microbe of acute osteomyelitis and with the form found in the metastatic pyæmic deposits.

But the acute infective periostitis of infants and adolescents differs entirely from the localised traumatic periostitis, such as occasionally follows a kick on the skin of a healthy adult at football, in that no characteristic micrococci are found if it goes on to suppuration, but slight superficial necrosis occurs at most, and no metastatic deposits are formed.

Secondly.—The *earliest* appearances of the disease are found in the *juxta-epiphysial* region.

[1] 'Fortschritte der Med.,' April, 1884.

[2] 'Revue d. Chirurgie,' Aug., 1885, pp. 636—663; and April, 1885, pp. 273 and 287.

[3] 'Abstract Med. Chronicle,' Sept., 1885.

[4] 'Archiv für klin. Chirurgie,' 1887, Bd. xxiv, s. 701.

With the object of testing the truth of this statement the writer made experiments on rabbits at the Pathological Institute in Leipzig, and was enabled by the kindness of the Council of the Royal College of Surgeons to examine the results.

The experiments and examinations were made in the years 1888 and 1889.

In the first instance a pure culture of the *Staphylococcus pyogenes aureus* was secured, and its efficacy was proved by repeated cultivations and final injection into the veins of a rabbit, the animal dying of pyæmia with multiple abscesses.

Experiment 1.—Black rabbit, aged six weeks. Under ether an incision was made down to the bone on the outer side of the right femur, and then with a carefully sterilised needle a puncture was made in the bone at the epiphysial line, and a pure culture of *Staphylococcus pyogenes aureus* in fresh distilled warm water was injected by means of a hypodermic syringe. A similar operation was performed on the lower end of the left femur, except that the microbes were injected into the medullary cavity. Both wounds were treated antiseptically and covered with iodoform and collodion. The rabbit died thirty-six hours afterwards.

Right thigh: No pus was found either in the soft parts or in the bone. The puncture was seen on longitudinal section to be in the juxta-epiphysial region one fifth inch above the epiphysial line, but not in the medullary cavity. There was much injection of the synovial membrane of the knee-joint, but the medulla showed no change.

Left thigh: At site of operation much staining of tissues with extravasated blood was seen. Medulla of the femur on longitudinal section was very red and diffluent, and presented several darkish purple spots which appeared to be local thromboses.

Microscopical examinations.—Sections through the right femur taken longitudinally[1] gave no evidence of any thickening of the periosteum around the site of injection and no formation of new cells in it; but the juxta-epiphysial region, especially

[1] A longitudinal section was made in all cases through the bones unless otherwise stated.

around the site of operation, showed Howship's lacunæ and osteoclasts.

The micrococci were especially abundant in this part, and arranged in groups.

Longitudinal sections of the left femur again show no change in the periosteum; but already, thirty-six hours after death, there were seen two yellowish areas in the section—one in the diaphysis near the growing line, and the other in the medulla of the diaphysis.

In both places the normal tissues were replaced by a mass of pus-like cells gradually fading off into the surrounding tissues (vide plate). In these masses numerous staphylococci can be seen with $\frac{1}{12}$ oil-immersion lens. It will be noted that in this experiment both periosteum and medulla came closely into contact with the septic fluid, and both were injured; but a change from the normal condition occurred, not in the periosteum, but in the medulla and in the young growing bone at the extremity of the shaft.

Experiment 2.—Rabbit, aged eight weeks. On June 13th an incision was made over the left external jugular vein, and it was exposed. With a carefully sterilised needle a dose of staphylococci was inserted into the lumen of the vein. The animal died June 20th.

Post-mortem examination.—Six hours after death extensive disorganisation of the right hip-joint; joint-cavity filled with pus, and an enormous collection of pus in the muscles around; also pus in left elbow-joint and an empyema in left pleural cavity; and abscesses in lung, heart, liver, and kidneys, but none in the spleen; left external jugular vein not thrombosed. On examining the various bones there was no pus beneath the periosteum; the medulla of the right femur and left humerus was much injected, darkish, and patchy, but there was no pus present. The ossific nuclei of the epiphysis were much inflamed.

Microscopically.—Right femur: In several sections is seen an area of broken-down bone on the shaft side of the epiphysial line. In this broken-down area are a number of rounded indifferent cells, probably pus-cells, and many micrococci. Beneath the periosteum no new cells and no signs of

infiltration with inflammatory material are evident. Another specimen also shows the same appearances, but the changes in the medulla are more marked. The left humerus also shows in the juxta-epiphysial region two yellowish areas which are fairly circumscribed, and the edges of which fade off into the surrounding healthy bone structure. These yellowish areas appear to be purulent foci, as the remaining small-celled material is stained bright red and not yellow when Orth's method of staining is employed, and another specimen[1] shows the same appearances in the same relative position.

Sections of liver stained by Gramme's method show minute scattered abscesses crowded with micrococci.

Experiment 3.—Young rabbit, age unknown. Under ether an incision was made in the lower end of the left femur, and the bone drilled and *Staphylococci pyogenes aurei* inserted as before. Into the opposite knee-joint a small quantity of the same micro-organisms was injected. Rabbit died twenty-six hours afterwards.

Post-mortem examination.—Rigor mortis well marked. On making a longitudinal section of the left femur the bone showed much injection, but there was no thickening of the periosteum nor effusion beneath it; the medulla was very red and in a semi-fluid condition, and in it were many darkish patches indicative of thrombosis. It may be interesting in this experiment to note again that in contrast to Experiment 2, and in similarity with Experiment 1, the rabbit expired much more quickly when the injection was made directly into the bone instead of reaching it through the blood.

Microscopically.—The right femur showed no advanced change. At the juxta-epiphysial junction the bony trabeculæ appear to be invaded and displaced by an ingrowth of new cells.

The left femur shows not only the point of drilling, but also numerous other fresh cavities, which are of a yellow colour and filled with a new material. This material on examination appeared to be composed of indifferent cells, roundish in shape and presumably pus-cells. The areas are well seen at the

[1] Other specimens stained by Gramme's method show the same changes—vide plate.

juxta-epiphysial junction, and there are some in the compact bone and in the medulla. In many of the yellowish areas in the latter part of the bone characteristic groups of staphylococci are seen with the $\frac{1}{12}$ oil-immersion lens. In other specimens there is seen a blurring and indistinctness of the columns of the proliferating cartilage at the epiphysial line. In no instance is the periosteum affected.

Experiment 4.—Rabbit, aged nine weeks. A quantity of pure cultivation of the microbe was inserted into the bony substance of the left femur just at the epiphysial line, as was afterwards proved. At the lower end of the left humerus, with a second and very carefully sterilised drill, a puncture was made deeply into its substance just above the elbow-joint, and the cancellous tissue broken up but no staphylococci inserted—in fact, every precaution was taken to prevent their entrance. The rabbit died five days after.

Post-mortem examination.—The left shoulder-joint was full of creamy pus, and in the muscles of the left thigh there was also an enormous infiltration. The bony nucleus of the upper end of the left humerus was inflamed and reddened, and so to a less degree was the lower epiphysis, but the medulla and cancellous tissue were of a dark red colour, the former softened in patches.

The microscopical examinations of the left humerus show no Howship's lacunæ nor osteoclasts; but in the juxta-epiphysial junctions are many broken-down areas of a yellowish colour, and in these very many micrococci are seen. These yellowish areas are assumed to be commencing foci of suppuration, both from the character of their contents and the manner in which they fade off into the surrounding healthy tissues. It is noteworthy that lining the inner surface of compact bone a layer of yellowish broken-down material is seen, formed evidently from the medulla. It may be perhaps advanced that if in this case the disease had progressed further, the bone would have died from cutting off of the blood-supply from within outwards, and not, as is supposed to occur in acute periostitis, from without inwards.

Experiment 5.—Rabbit, aged eight weeks. An implantation

of staphylococci was made at the lower end of the left femur in the following manner:—The periosteum was carefully separated for a considerable extent off the bone, and then beneath the periosteum a mass of staphylococci from a pure culture was inserted, and the parts replaced as carefully as possible, the wound closed and dressed antiseptically.

The rabbit was killed in two days' time, and on examining the left femur there was no effusion, no pus beneath the periosteum, and it had even become adherent to the bone again. The only change (naked eye) produced was some reddening at the point of puncture. On making a longitudinal section of the bone, the cancellous tissue and medulla showed no striking change from the normal.

Microscopical examination of the lower end of left femur.—The periosteum at the site of implantation of the micrococci appears in no way to be separated from the bone; and although it appears to be thickened, yet the writer thinks that such thickening is not greater than the natural conditions where the periosteum divides, one portion to pass downwards to the joint and the other inwards to the epiphysial line.

The medulla shows certain areas in which numbers of ill-defined cells teeming with micrococci are seen, and at the juxta-epiphysial junction the normal line of bone proliferation is obscured by a granular material, and in some specimens these masses have taken the iodine stain very deeply when Gramme's method has been employed (vide plate).

It is, therefore, more than probable that although in some instances the microbes may obtain an entrance into the bone through the blood-vessels of the periosteum, yet the actual site of their development is in the unstable and rapidly growing vascular tissue in immediate proximity to the epiphysial line, and pus formed there makes its way outwards to the periosteum.

Experiment 6.—Rabbit, aged seven weeks. The left tibia was fractured on June 26th, and on June 28th an injection of staphylococci was made into the left jugular vein. The animal was killed on June 30th, but no matter was found at the site of fracture, although there was an unusual amount of inflammatory effusion seeing that the fracture was transverse. (This

effusion is explained by the absence of apparatus required to keep the fragments in position.)

Microscopical examination.—Results nil—except that one can see a few cartilage-cells in the callus, such as are described as occurring in the union of fracture in animals.

Experiment 7.—Rabbit, aged five weeks. The left tibia was fractured on June 28th, and an injection of staphylococci was made into the blood on June 30th. The animal was killed on July 6th.

Post-mortem.—To the naked eye the change did not appear more marked than in the preceding case.

Microscopically.—Specimen shows a large amount of effusion around the periosteum, consisting of numerous indifferently shaped cells, some of which are seen to be changing into fusiform cells, but there is a striking absence of purulent cells and micrococci.

Another specimen shows vacuolated areas in the medulla. These are perhaps due to the intercurrent inflammation produced by the fracture. But in the medulla many microorganisms are visible. On the whole, the changes in the direction of increase of tissue are chiefly demonstrable in the periosteum, owing to inflammatory effusion.

From these experiments we may say with some reason that of the medulla, juxta-epiphysial junction, and periosteum, the first two are more susceptible to the presence and irritation of the specific microbes, and are the earliest to take alarm; while the periosteum is so comparatively tolerant of them that although a mass be implanted beneath it as in Experiment 5, yet it shows no striking departure from the normal condition for at least two days, while already the medulla and juxta-epiphysial regions are undergoing morbid change. In Experiments 1, 3, and 4, the periosteum was injured yet no separation occurred, and no formation of pus, but purulent foci were found in the marrow and young growing bone at the end of the shaft.

Inasmuch, then, as the juxta-epiphysial junction is in these experiments the site of early inflammatory change, it is but reasonable to expect that if the animals had lived long enough

we should have found from the anatomical relations of the parts an acute purulent periostitis and osteomyelitis.

The question why acute infective bone disease is so prone to occur at puberty must be left unanswered at present.

It is sufficiently easy to speak of unstable tissue, tissues more or less embryonic, or in course of development; but is not the whole body in the same condition of rapid growth?—and why the virus should attach itself more firmly to one growing tissue than another is still an open question, and demands further investigation and experiments.

Thirdly.—In cases of necrosis of the diaphyses the destructive process is most marked at the extremities, the bone being found eroded and often gangrenous; and further, there is such a loss of tissue that the dead shaft no longer fits accurately into the epiphysial and periosteal cap. "There can be little doubt that inflammation in bone occupies seats of election which are those of greatest physiological activity. The inflammatory disposition is here, as in all other tissues, in direct ratio to the proliferation of the anatomical elements;"[1] for example, witness the extreme susceptibility to morbid and rapid change, under sufficiently powerful stimuli, of adenoid tissue, such as the tonsils in tonsillitis, Peyer's patches in typhoid fever, and many other lymphatic organs where the production and destruction of that marvellous white corpuscle, the probable centrum of all pathological processes, is continually carried out.

Klose, in describing the condition of the parts in his case of meningo-phlebitis, which is now held to be acute infective periostitis, speaks of the extremity of the diaphysis being found in a roomy cavity bare of periosteum and dark in colour. The bone is surrounded by pus which has an ichor-like character. At the point of junction between the epiphysis and diaphysis the latter is spongy and soaked in pus, and the line of union is marked by a more or less deep groove where the division between the parts will occur.

The longer this stage of disease is continued the more is the end of the diaphysis softened, and the deeper is the groove around it.

The pus has separated the periosteum a long distance off

[1] MacNamara, op. cit.

the shaft of the bone, and later on perhaps perforation of the epiphysis occurs, with destructive arthritis ensuing.

Reference to some cases will serve to emphasize the exceeding amount of inflammatory destruction of the shaft at its extremities, and the writer thinks that such destruction limited to the extremities points not only to the nature of the morbid process but to its actual site of origin and focus.

Mr. Jonathan Hutchinson, jun., published in the 'Annals of Surgery,' vol. xi, p. 96, the following case of Mr. Treves.

A child, æt. 6, after an injury to the left arm, due to a fall out of window, was admitted into the London Hospital with great swelling of the hand, forearm, and arm. It being evident that suppuration had occurred, several incisions were made and pus was evacuated containing many micro-organisms, but the patient died subsequently of pyæmia. At the autopsy suppuration was found to have occurred in the elbow-joint, and the upper epiphysis of the radius was lying loose in the joint-cavity; the periosteum peeled off the radius, especially at the upper part of the shaft, and on section the neck of the radius was seen to be intensely inflamed.

Mr. MacNamara, in the appendix to his work on 'Diseases of the Bones and Joints,' publishes notes of the case of A. L—, æt. 14, who received an injury to his right knee four months previously. Eight days after admission, as it was evident that the boy was suffering from acute epiphysitis of the upper end of the right tibia, an incision was made and pus let out, but, the patient showing symptoms of septicæmia, sequestrotomy was performed. The shaft was found separated at its upper end, but the lower extremity was still attached to its epiphysis, so the bone was sawn through. On section of the shaft longitudinally the medulla contained in the cancellous tissue of the upper half of the shaft was found in a state of acute inflammation, and the upper surface of the diaphysis was thickened and necrosed. The smell proceeding from the cut surface of bone was most offensive. The patient subsequently recovered.

Another case, quoted by Dr. Fagge[1] apropos of the diagnosis of acute rheumatism, and pyæmia arising from acute periostitis, showed at the autopsy inflammation of the lower part

[1] 'Principles and Practice of Medicine,' 1st edit., vol. ii, p. 550.

of the shaft of the right femur and adjacent epiphysis with denudation of the periosteum.

Another case was under the care of Mr. Bryant in Guy's Hospital.

A girl, æt. 17, was admitted suffering from necrosis at the lower end of the femur. On operating, a sequestrum 1½ inches long, gangrenous, and foul, was removed from the lower end of the shaft.

All these instances demonstrate the peculiar violence of the inflammation in the juxta-epiphysial region, while in nearly all the periosteum appears to have remained attached to the shaft, and not to have been so profoundly affected as the juxta-epiphysial junction.

Specimens illustrating the ulceration at extremities of the shaft are found in the Hunterian Museum: 1218 C, a section of the upper extremity of the shaft of a humerus which has been inflamed and separated from its epiphyses, and its upper surface is seen ulcerated; 1218 B, the specimen removed from Mr. MacNamara's case (p. 94); Guy's Hospital Museum, 1155[30], epiphysitis of the great trochanter with diaphysitis in a child —specimen shows a large abscess-cavity occupying and extending on either side of the epiphysial line.

Many other specimens of acute necrosis also show the grooving of the end of the shaft in cases of separation.[1]

From these and other specimens it is but fair to assume that in some of them it was just as probable that the disease commenced at the junction of shaft and epiphyses, as that it commenced beneath the periosteum; and in others the balance of evidence is largely in favour of the disease commencing in the former position rather than in the latter, seeing that in the juxta-epiphysial junction it was more advanced and greater destruction of tissue had occurred. Another point in favour of this is that if necrosis gives rise to fracture in adults it occurs not as a rule near the end of the bones, but near the middle, while in children fracture almost always occurs at the extremities of the diaphysis, these being the parts of the bones most subject to disease which cuts off their blood-supply.

[1] St. Bartholomew's Hospital Museum, Nos. 31A, 103, 67; London Hospital Museum, No. 610; University College Hospital Museum, Nos. 475, 456; Guy's Hospital Museum, No. 1239.

That the disease is most intense at the line of growing bone is borne out by another consideration. In instances where the joint becomes implicated by extension of inflammation or irruption of pus into it, these events occur almost invariably by perforation through the epiphysis and not by extension beneath the periosteum. The writer can only quote one museum specimen where disorganisation of the joint occurred from the latter cause, and that is No. 604 in the London Hospital Museum. If, then, the disease be an acute periostitis purely and simply, should we not expect it to attack the epiphysial as well as the diaphysial periosteum? Yet how common it is to find the living epiphyses surmounting a dead shaft!

It may be objected that only an acute inflammation beneath the periosteum spreading into the juxta-epiphysial junctions would entail total necrosis; but in answer to this it is equally fair to assume that pus may be formed at one extremity of the shaft, and then, passing beneath the periosteum up to the other extremity, separate it from its epiphysis.

Mr. Jonathan Hutchinson, jun.'s case (p. 94) shows that such might have been the course of events had the patient lived longer.

In the records of necrosis of growing bones we find all grades of it, but one particular variety to which it is necessary to allude is that in which the necrosis is strictly limited to the diaphysis and to the end of it, while the changes in the neighbourhood are by no means so far advanced and the periosteum has suffered but slightly. A series of such cases was published by Mr. Eve in the 'St. Bartholomew's Hospital Reports,' vol. xx. The cases were briefly—

1. Necrosis confined to the extremity of the lower end of the shaft of the femur, causing separation of the epiphysis with destruction of the knee-joint. About three quarters of an inch of the end of the diaphysis were found necrosed.

2. Necrosis of the extremity of the diaphysis and in the upper extremity of the tibia, followed by disorganisation of the joint, in a boy aged two and a half years. After amputation, a piece of necrosed bone the size of a walnut was found at the extremity of the diaphysis.

3. Necrosis of the lower end of the tibia in a girl aged fourteen, following a sprain. Amputation was found necessary,

and, on making a section through the lower end of the tibia, a cavity large enough to contain a walnut was found at the extremity of the diaphysis completely filled up with granulation-tissue.

The epiphysial line was also extensively eroded.

Cases of chronic abscesses of bone which are universally admitted to occur more commonly at the ends are the sequelæ of acute or subacute limited ostitis, and from their position point to the more common site of inflammations of growing bone—in the juxta-epiphysial region.

Fourthly.—If the origin of the disease as a juxta-epiphysitis be conceded on the foregoing grounds, then a more thorough and radical method of treatment is necessary; and operators must not merely be content with incising the periosteum, but should in all cases attack the disease at its starting-point by tunnelling the juxta-epiphysial junction, and so allow a sufficient exit for the products of the inflamed medulla and cancellous tissue.

In the most recent work, 'On Diseases of the Bone,' Mr. Jones, speaking of osteomyelitis, says: "The great desideratum is the early recognition of these cases and the prompt adoption of some simple method of treatment, such as free periosteal incisions with removal of the suppurating marrow through trephine holes" (p. 84); but, writing in an earlier part of his work (pp. 37 and 38), he remarks: "Acute periosteal abscess has to be diagnosed from acute diffuse cellulitis and acute osteomyelitis. The age of the patient, the character of the pain, usually limited to the diseased bone, together with the effect produced by exploratory incisions, will help in discriminating it from the former; while the *course of the disease and the insufficiency of incisions through the periosteum alone*[1] are points to be remembered in distinguishing it from the latter." Further in speaking of the diagnosis of the nature of the bone disease, he adds: "The *subsequent course* of the disease will give valuable information as to whether it is periosteal or endosteal."

Now Ollier[2] states that "trephinings made antiseptically into bone do not produce necrosis, they constitute on the

[1] The italics are the writer's.

[2] 'Traité des Resections et des Operations.'

contrary the best means of preventing it." Trephinings into inflamed long bones are attended with no more risk than trephinings of the flat bones of the skull when the pericranium and subcutaneous tissue are inflamed.

Why then wait until the course of the disease has enabled the surgeon to complete his diagnosis? By that time in all probability the disease will have caused partial or entire necrosis of the shaft, and grave constitutional symptoms will have set in. Surely it is better, even in the early stage of the disease, when there is marked pain, associated with swelling and fulness of the veins of the limb and rigors, to make a single incision down to the bone through the soft parts and periosteum, and then trephine in the juxta-epiphysial region, and at a point about an inch higher up, and so open up at once all three structures—the periosteum, the juxta-epiphyseal junction, and the medulla. Should improvement then not set in, it would not only be permissible but rational to open up the medullary cavity at the other extremity of the diaphysis and clear out the medulla.[1] Such a course will give the affected bone the best opportunity of recovering itself, and the patient will be spared not only the immediate dangers but the more prolonged ones arising from long-continued suppuration due to exfoliation of the necrosed tissue with formation of new bone.

Mr. Bryant[2] as long ago as 1879 was of opinion that "a free incision down to the bone in periostitis as well as in endostitis does nothing but good, and drilling or trephining inflamed bone, even when it fails to cure, tends to check the progress of the disease, and relieves pain."

The writer then would urge most strongly that in all cases of acute bone disease of young subjects the treatment is incomplete without at least trephining the juxta-epiphysial junction. Such a course can have no possible effect on the subsequent growth of the bone so long as the aperture is made above or below the line of proliferating cartilage; and its early adoption can only be attended with such good results, that removal of the entire shaft by sub-periosteal resection will no longer be called for, nor is the neighbouring joint so

[1] Cf. successful case by König, 'Centralblatt f. Chirurgie,' 1880, No. 14.

[2] 'Guy's Hosp. Rep.,' ser. iii, vol. xxiv, page 17.

likely to become affected either by perforation of the epiphysis, or by extension along the periosteum.

Fifthly.—The channels of ingress of the micrococci.

They may and probably do find an entrance into the blood through the respiratory tract, and Kraske,[1] notices the not unfrequent occurrence of pneumonia in advance of bone disease. Some, too, will find an entrance through the digestive tract; but it appears that we must look rather to the skin as the main portal of the organisms. Some personal observations here may throw light on the matter. In examining sections of skin from a case of this nature the sweat-ducts were seen to be more dilated than usual and to be crowded with micrococci.

Now, osteomyelitis and acute periostitis occur in those very bones which are more exposed to violence and least covered by the tissues. It seemed from this fact that the usual history of a slight blow has more significance than is at first apparent. The writer has observed that the skin in bruises presents one of two appearances—it is either excessively dry or else covered with a copious perspiration, pointing respectively to occlusion of the minute subcutaneous arterioles and capillaries by extravasation, or to paralysis of the vaso-motor nerves. In the former condition of dryness of the skin it is very possible that some micrococci present on the surface of underclothing may be rubbed into the sweat-ducts and there proliferate and so make their way to the bone. It is advantageous to couple with these observations Kraske's remark that in many cases, inflammation of bone follows and is consequent upon a primary inflammatory focus in the skin and subcutaneous tissue and this has also be noted by Lannelongue and Pettit, independently. Rosenbach's experiments in producing osteomyelitis with pus from boils have been frequently confirmed.

It is desirable to obtain experimental confirmation of the part the skin plays, and the writer thinks this might be effected by the administration of belladonna to rabbits, rendering the skin dry, and then rubbing in the micro-organism in distilled water, and further confirming any results so obtained by bruising the skin over the lower end of the femur of another

[1] Quoted by Sajous, 'Annual of Med. Sci.,' 1889, vol. ii, p. 286.

animal and applying the micrococci in the same way, noting the effect on the bone and microscopical appearances in both.

The share taken by the periosteum in the increase of the total weight of the bone cannot without difficulty be compared to that taken by the epiphysial line; but presumably the share of the latter is much greater, remembering how much greater is the increase in the length than the thickness of bone. Physiological activity is greater at the ends of the shaft than at its circumference if we judge only from the nature of the bone formed. At the extremities soft cancellous bone is rapidly produced, while at the circumference hard compact bone is formed. The blood-vessels are larger and the circulation is slower in the former position; and that deposition of the micro-organisms should occur more readily in the juxta-epiphysial junction than beneath the periosteum is what might reasonably be expected.

DESCRIPTION OF PLATE.

Illustrating Mr. Tubby's paper on Acute Infectious Periostitis and Acute Necrosis of Growing Bone.

This plate shows a longitudinal section of the lower epiphysis of a rabbit's femur, stained by Gramme's method. The yellowish areas in the juxta-epiphysial region are due to foci of suppuration following the inoculation of *Staphylococcus pyogenes aureus* (pp. 88, 89, 91).

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CHIEFLY CONCERNING BRUITS.

A Lecture on Pathology from a Course upon "Points in the Pathology of Diseases of the Heart."

By JAMES F. GOODHART, M.D.

This lecture formed part of a course of lectures on the pathology of heart disease delivered at Guy's Hospital in the summer of last year. I had intended to recast it and make it special to diastolic bruits in the great vessels, but time has failed me, and on looking it over in order to excise such parts as are foreign to my particular intention, I find the lecture does not admit even of this.

It is therefore published as it was delivered, and I must beg my readers to remember what the paper is—a lecture; to forgive the elementary statements that it contains; and to try to pick out the few crumbs of thought and fact within it. I shall even hope that as it is, it will not prove altogether uninteresting.

The special points to which attention is directed may be thus summarised:

1. Stenosis of the valves is slow in maturing because of the dilating and hindering force of the blood-stream.

2. The frequency of presystolic bruits on the left side and their rarity on the right side; and this out of proportion to the admittedly rarer occurrence on the right side of the conditions requisite for their production.

3. The occurrence of diastolic bruits in the great vessels of the thorax not dependent upon valvular disease.

4. The frequency and clinical importance of dilatation of the pulmonary artery.

5. Its adequacy to explain many of the so-called hæmic murmurs.

6. The inefficiency of our means of diagnosing simple hypertrophy of the left ventricle.

7. The frequency of simulated hypertrophy of the left ventricle, due to displacement of the impulse by hypertrophy and dilatation of the right side of the heart.

The subject under consideration is malformations of the heart. As might almost be imagined from the seeming complexity of the process of the formation of the aorta and pulmonary artery, the malformations, other than those at the arterial ostia, are rare indeed. And here it occurs to me to say, since I have introduced the question of evolution and design, that our good dame Mother Nature is not always equal to the intricacies of the situation she has created for herself; and from the opposite point of view to that advocated in the last lecture this may very fairly be used as an argument against the existence of the very special control over our development advocated by some—for if it were so, how could these mistakes occur?

There are many forms of malformation—patency of the foramen ovale; patency of the ductus arteriosus; deficiency of the septum of the ventricles; a single ventricle and auricle; a single ventricle to the double auricle; adhesion and stenosis of the various valvular orifices, chiefly of the pulmonary artery and of the aorta, less commonly of the tricuspid and mitral, either singly or in company; and then there is transposition of the viscera. But the only ones that you will meet with any frequency, almost the only one you will meet with on the shelves of our museum, will be the more or less complex one of communication between the two ventricles, stenosis of the pulmonary artery, and a patent ductus arteriosus. I say *more* or *less* complex, because sometimes the pulmonary artery is quite obliterated, sometimes only much contracted; sometimes the ductus is closed, sometimes not; sometimes the foramen is open, sometimes it

is not. Patency of the foramen ovale; patency of the ductus arteriosus; thickening and stenosis of the tricuspid and mitral valves; and thickening and stenosis of the aortic valves—of these various conditions let me say a few words.

As an out-and-out malformation the first named is by far the most common; Dr. Peacock found it, or at any rate some obstruction or other to the passage of the blood from the right ventricle or main pulmonary artery, 119 times out of 181 cases of malformation. Kussmaul found that of ninety cases twenty-six were cases of atresia or narrowing of the pulmonary artery. It is usually narrowed as far as the bifurcation, and in 13 per cent. the ductus arteriosus is closed, suggesting, as Dr. Peacock pointed out, that this malformation is due to an imperfect development of the branchial arch on that side. The pulmonary artery may have very thin walls, or be quite stunted, and transformed into a mere cord. The conus arteriosus of the right ventricle is occasionally narrowed, the sinus being hypertrophied and dilated. The aorta is usually dilated, and springs from both ventricles, but its relation to the pulmonary artery is normal. The size of the aperture in the septum is subject to much variation; the foramen ovale has been found open in about three fourths of the cases. The ductus arteriosus is sometimes open, but more often closed. But on the whole it is common to find, under such circumstances, that the pulmonary valves are adherent, thick, and contracted, the pulmonary artery very small; the septum is deficient, and the aorta seems to arise sometimes more, sometimes less, from the right ventricle, corresponding with the more or less of deficiency of the septum. It is also not uncommon to find in these cases that the tricuspid is thick, and has vegetations upon it, and the mitral and aortic valves may be thick also. And these additional changes are of great moment and interest in relation to the general pathology of valvular disease, as I shall show you by-and-by. The right ventricle is usually much hypertrophied, and the heart assumes a squarer shape than natural in consequence of this.

Now this complex condition is one that has been much discussed, much written about, and, it may be added, much obscured in consequence. Some people hold that the whole

thing is purely one of arrest of development, which suggests all sorts of possibilities for the exercise of an active ingenuity; but it is an explanation that offers but a scanty meal to one who would feed upon something definite. Others give an explanation which on the whole satisfies the conditions, and explains them in a manner that is sufficiently reasonable to be easily remembered. An explanation of that kind in any branch of inquiry, although not incapable of detraction, always seems to me to possess a strong probability of being correct. It is this that in the early stages of development—and how early this is, and therefore how little we can know about the conditions, may perhaps be better appreciated when I ask you to remember that the semilunar valves are formed and well pocketed in the chick about the sixth day of incubation, and at a relatively early period in mammals—something goes wrong with the development of the pulmonary valves, or to the conus arteriosus, and the blood is unable to pass from the right ventricle along the pulmonary artery to the ductus arteriosus. If the septum be still incomplete between the ventricles, the obstructed current will necessarily pass through the hole in the septum to reach the aorta, and the septum will thus be prevented from closing. I might stop here to note, as I am fond of doing, how much the history of valvular disease is a departure from or modification of the doctrine of cicatricial contraction in consequence of the dilating force of the blood-current, but there will be other and better opportunities of doing this later on. But I will say this, that the history of valve lesions affords strong evidence of the probability of this explanation, that the blood-current passing to the ventricle does actually keep the septum open. Well, then, if it keeps the septum open, and the blood passes in any quantity, the current will naturally tend to modify the position of the great vessels of the heart, and to mould the aorta more to the right, so that, as I have said, it is common to hear these cases spoken of as the aorta arising from both ventricles, or even more from the right than the left.

There are other cases in which the vessels are transposed, or so much displaced as to make them clearly malformations; but the more common affection, that in

which the aorta is situated over the septum more to the right than it should be, is, I agree with Dr. Peacock, quite adequately explained by the action of the disturbing force of the blood-current shot through the imperfect septum into the aorta.

For some reason or other, it is hard to say why, congenital malformation of this kind is almost always accompanied by considerable thickening of the valvular structures. One explanation, and a very probable one, is that the malformation, by interfering with the natural and easy current, causes increased work and increased wear and tear, and therefore thickening.

Further than this, it is by no means uncommon to find the valves thickened and fringed with recent vegetations, showing that they are prone to more acute inflammatory changes, and there are numerous specimens of this fact upon the shelves of our museum.

One would suppose, from such a variety of circumstances, that the physical signs would be likely to vary in the different cases. And so no doubt they do, but on the whole I think it best to say, as more conforming with what will, I believe, be your experience hereafter, that it is remarkable how little they do vary. It is, I think, not improbable that to a large extent the change in one valve may modify the conditions under which bruits might be expected to be, and would generally be, produced in some other, and thus lead to what is in all these cases the most common feature, that of a simple rasping, roaring, systolic bruit, heard all over the precordial area, and not uncommonly in the back also. But all the changes I have mentioned may be present and no bruit of any kind audible, cyanosis alone being the evidence of the existence of the disease.

This condition is compatible with many years of *existence*—I won't call it life. Naturally, the less the malformation the more the chance of a longer life, although existence is sometimes wonderfully prolonged under conditions which would seem to be most adverse.

Of patency of the foramen ovale there is little to say, although, if I remember rightly, it is the one malformation of which we hear most in our earlier anatomical studies.

But it is one which is relatively unimportant for two reasons: first, because there is no constant symptom by which it can be recognised; and secondly, it is not so very uncommon to find it in the post-mortem room, when its presence has never been suspected during life. It co-exists often with other malformations of the heart, but alone its equivalent of disturbance is apparently an inconstant one. And perhaps in this regard it may be suggestive to couple it with the fact I have just alluded to, viz. that there may be a largely deficient septum between the ventricles, and yet no bruit. Bruits come about under a variety of circumstances, but they mostly depend for their production upon the muscular propulsion of the blood-stream from one cavity into another by some aperture, which is considerably smaller than the cavity into which the blood is driven. Obviously the conditions are fairly parallel, though they differ in degree in the case of auricles and ventricles; and if a bruit may be absent even under such favorable circumstances as the strong driving power of the ventricle, *a fortiori* is it likely that there will be a more frequent absence of bruit when the muscular driving power is so much more feeble as is that of the auricles.

I take it that this fact is well illustrated in the difference that is known to exist in the two auricles in the matter of the auricular systolic bruit. You know how often you hear these bruits on the *left* side of the heart, and how seldom, I might say never, they are heard on the right side. The explanation that sufficed for a long time was, that what was common on the left side was very rare on the right, and that consequently not being on the look-out for the exceptional we missed it when it occurred. But this is not so as far as the rarity of contraction of the tricuspid is concerned; for although admittedly it is far less common than the same disease of the mitral, it is not so uncommon but that it might be diagnosed several times in a year in a large hospital like this if the disease were accompanied by a recognisable bruit. But in any case the existence of a bruit depends upon such nice questions as muscular fitness, balance of power, the relative size of the apertures through which the blood passes, the condition of the surfaces over

which the blood passes, &c., questions which it is often impossible to say much about during life, and hardly less so after death—that they are perhaps more interesting to think about and to attempt to unravel each for himself, and in each case more or less for itself. But of this more directly.

Nor should I have said much concerning simple patency of the ductus arteriosus had it not involved a practical matter such as the production of murmurs. It possesses more a clinical interest than a pathological one, for it would appear to be sometimes associated with a bruit which is not only of a peculiar quality but also of unusual time. Its quality is harsh and humming, its time diastolic but difficult to gauge accurately, it is so long, and may obliterate the second sound. In thus describing the bruit I must confess that I am in some degree following a common and bad practice of many writers of books, and drawing more upon my imagination than upon fact. Facts, indeed, are so scanty that in looking over six standard treatises upon diseases of the heart I can find only one case, and that repeated with monotonous reiteration in all of them; and this case is reported by the late Dr. Hilton Fagge. Dr. Fagge describes the case thus:—Talking of diastolic bruits, he says, "Another infinitely rare condition in which a diastolic murmur may be heard is that in which the aorta communicates with the pulmonary artery either by a patent ductus arteriosus or through an aneurismal sac. The murmur was in part musical, was audible at the second left costal cartilage, and was transmitted to the left along the cartilage, but not downwards along the sternum. It was not everywhere continuous with the second sound. It had a wavy character, quite unlike anything I had heard before." Dr. Fagge adds that in its main features the bruit in this case resembled that heard in a case published by Dr. Wade of Birmingham, and in which a communication between the aorta and pulmonary artery was diagnosed in consequence, and an aneurism between the two found after death. The points insisted upon are the diastolic rhythm, prolongation of the bruit nearly to the commencement of the next cardiac systole, and its inaudibility at the apex of the heart. I feel

inclined to add to this description that I believe a *systolic* bruit, rather *late* in its commencement, may also be produced under these circumstances; and I think so for the very reason indicated by Dr. Fagge, that aneurismal communication between the aorta and pulmonary artery provides a somewhat similar state of things, and I should say sometimes gives a continuous bruit in part occupying the systole, in part the diastole. It so happens that I have had a rather exceptional experience in cases of this kind, and in no less than five cases has it been possible to make a diagnosis from the peculiar character of the bruit. Naturally it is by no means always of quite the same quality, but the sum of my experience is that it is a little late in the systole in commencement. It is peculiarly harsh, continuous, and probably musical, and I should be inclined to add that the commencement of the bruit at the commencement of the systole, or a little late, depends upon the amount of the dilatation of the receiving vessel and the size of the aperture between the two vessels.

For teaching purposes, however, let me say that on a review of the few cases that have been recorded and that I have seen in which the morbid conditions have been somewhat similar, and in which the bruits produced might have been expected to resemble each other more or less closely, it is clear that the abnormal sound has sometimes been systolic and sometimes diastolic, and the reports of the cases do not allow of any distinction being founded upon the time of the bruit, but that peculiarities for distinction have rather existed in the quality of the sound and in the position in which it has been heard. One of the last cases of this kind that has occurred to me I have upon the table. In it it may be seen that the aneurismal sac is in a somewhat unusual position, being very much in the site of a dilated ductus arteriosus. It occurred in a man of forty-six, who had been in the army, and had had syphilis. He was admitted with extreme dyspnœa, absence of entry of air in the left lung; there was a loud, harsh, grating bruit at the base, systolic in time and loud over the aortic valves, but becoming bell-like and musical as the pulmonary valves were reached, and a coarse thrill was felt here. The bruit was much less audible at

the apex. From these physical signs I predicted the existence of an aortic aneurism which had effected a communication with the pulmonary artery, and such was found to be the case. At the same time that this case was in the ward another man lay opposite him with a peculiarly harsh, double aortic bruit, and in the third left space a distinct continuous thrill, described in the report as resembling an arterio-venous one, and over the same area a systolic bruit followed by a sound of rumbling character. I felt sure from the character of the sounds that we had to do with an aneurism of the intra-pericardial portion of the arch, and also that the sac had formed a communication with the pulmonary artery, and in this case too the predicated conditions were found post mortem. Well, then, having said thus much, I must immediately reduce the value of whatever of positiveness there may be about this statement by pointing out that a patent ductus arteriosus—a narrow pipe between the aorta and the pulmonary artery—is not a similar condition to an opening directly from one channel into the other, nor as a sac between the two, such as I have just shown you. Clearly the facilities for the passage of blood from one vessel to the other, and therefore for the production of a bruit, are much more pronounced in the one case than in the other. And further than this, I am, for my own part, very much inclined to doubt, although the ductus was patent in Dr. Fagge's case, whether the bruit heard was produced at the tube, because I have heard a very similar bruit—at least, so I suppose from Dr. Fagge's description, but the description of a sound is a very doubtful affair at best—in what ultimately proved to be a simple but enormously dilated pulmonary artery. This case—chiefly under the care of the late Dr. Moxon, but in his absence frequently under me also, was that of a man aged fifty-three. He was in the hospital for a long time—from February till June, in fact,—and during that time I frequently saw him and discussed the nature of his ailment from the bruits that were heard. There was some difference of opinion as to the nature of the case. His history was well calculated to put us on the wrong tack, as it often is. He said that he was quite well till one day, six years before, he was walking up St. Michael's Mount, when something gave

way in his chest and he was obliged to stop, and he had been short-winded ever since. On admission the precordial area was large, and there was a peculiar, not very distinct, wavy impulse in the second left interspace. Over this area a corresponding long diastolic musical bruit was heard. The bruit obliterated the second sound, occupied the whole of the diastole, and seemed to me to be most probably dependent upon an aortic aneurism, which might not improbably communicate with the pulmonary artery, from the humming character of the sound, its length, and its obliteration of the second sound. But it turned out to be what nobody had, I think, suspected, viz. a simple dilatation of the pulmonary artery dependent upon chronic obstruction in the lung from fibroid changes and emphysema, the sigmoid valves being healthy.

Now you will notice that this case is practically a large fusiform aneurism of the pulmonary artery, and somehow or other a diastolic bruit was produced in consequence. And I notice in Dr. Fagge's case that the heart weighed 22½ oz., the left ventricle was dilated and hypertrophied—a condition ascribed to the renal disease which was present; the right ventricle likewise; the right auricle was greatly dilated, much more so than the left; its appendix was opened out, and its enlargement might fairly be called extreme; the endocardium of the right auricle was opaque. The tricuspid orifice was much enlarged; the muscular substance of the right ventricle was hard and tough. *The base of the aorta was much less in size than the base of the pulmonary artery, and the two primary divisions of the latter vessel were greatly dilated, especially the right one, but the coats of the pulmonary artery were apparently not thickened.*

It is, of course, possible that this condition of great dilatation of the pulmonary artery in Dr. Fagge's case might have been engendered by the abnormal passage of blood from the aorta into the pulmonary artery. I am quite inclined to think that abnormal currents and vibrations may have some influence in demoralising the nervous control, and in paralysing the muscular action of the ventricles, and thus of favouring dilatation. Quite possibly abnormal vibrations may have some similar detrimental effect on the pulmonary *artery*, for neither is it beyond the range of nervous

influence. But what I want to insist upon is that the state of the lungs, the pleuritic effusion, the disease of the left side of the heart, are in themselves quite sufficient to explain a dilated state of the pulmonary artery, so great must be the obstruction thus produced to the passage of blood through the lungs; and the dilated pulmonary artery is in itself quite sufficient, on the evidence of the case just detailed, to explain the occurrence of such a bruit as that which was audible in Dr. Fagge's case. Therefore the evidence in favour of the production of definite and diagnostic physical signs in cases of patent ductus arteriosus is flimsy indeed, seeing that even the single case is open to some question; although I admit that, adopting the fluid vein theory of the production of bruits, we have apparently in a patent ductus the most favorable condition possible for the production of a murmur.

Now, before reverting to the subject of malformations, I shall take the opportunity which this short discussion of the causation of one special bruit offers of diverging for a few minutes into the subject of bruits in general. This is a question of physics rather than of pathology, but clearly it is not easy to separate between the morbid changes themselves and the physical signs to which they give rise. The theory of the causation of bruits most adopted at the present day is that I have just alluded to as the fluid vein theory. Fluid passing from a narrow channel into a wider one, or from a narrow aperture into a *cavity*, is thrown into vibrations, and a murmur is produced in the wider part of the vessel. One must not go so far as to say that this explanation applies to all murmurs; but it is very generally applicable both to bruits in the vessels, and also to those in the heart. In the aorta a systolic bruit will be produced when from any reason the valvular orifice is contracted, or the canal itself is dilated; or when from the canal a sac juts out in one place or another, its orifice being somewhat contracted. In the pulmonary artery, in the same way, contraction of the orifice of the artery by disease of the valves leads to a bruit, and so also does the dilatation of the artery that destroys the relation between the orifice and the canal. A diastolic aortic bruit will thus be produced by anything that

renders the valves incompetent; and, other things being equal, will be all the louder, the greater the disproportion between the aorta and the ventricle—that is to say, the more the dilatation of the ventricle. Then take the auriculo-ventricular orifices. If they do not close accurately there will be a septum which throws the blood into vibrations as it passes through the valve into the auricle, and often a widely dilated auricle, beyond it—and thus a bruit. Supposing the auriculo-ventricular orifice contracted, and blood passing from the auricle into the ventricle, again there will be the conditions for the production of a bruit; while still further, supposing that the ventricle be widely dilated, as is often the case in mitral regurgitation, blood forced into the auricle during systole will cause a systolic bruit as it passes into the auricle, and a diastolic one will be audible as the elastic and muscular recoil of the already over-distended auricle sends its systolic charge back again into the ventricle during the diastole.

For the veins a similar explanation holds good. Murmurs are produced by irregularities in the calibre of the canals along which the blood passes; and in anæmia, the blood being poor and scanty, the veins contract upon the diminished blood-stream where possible, but about the root of the neck, held out by virtue of their anatomical position and the structures that surround them, contraction is not possible; and hence, it is supposed, is produced a series of contractions and dilatations which fulfil the conditions necessary for the production of murmurs.

With reference to the pulmonary artery, I want to point out that not much about it is to be found in books. You will hear a good deal about accentuated second sounds over the pulmonary artery, as an indication of the retardation of the blood-stream in the pulmonary circuit, and be taught in great measure to gauge thereby the amount of damage, or failure of compensation, that is accruing from disease of the left side of the heart, but I think morbid anatomy takes us further than that. From post-mortem observation I believe it can be shown that dilatation of the pulmonary artery is no uncommon occurrence, although I will add, as a caution, it is one which is overlooked in making an exa-

mination with great readiness. I had had my attention drawn to this point more particularly by the case that I have already dwelt upon; and since then I have seen several cases. In one of these, a case of bad bronchitis, a blowing diastolic bruit was heard to the left of the sternum, which was presumably produced in the pulmonary artery; and at another time an indistinct bruit is said to have been attached to the first sound, and there was a harsh thrill over the cardiac area. The practical point of this is that if the pulmonary artery will dilate readily, the causes of pulmonary retardation being legion, a reasonable *a priori* argument in favour of the bruits which occur so frequently in the precordial region corresponding to the pulmonary artery being really produced in that vessel is afforded. I need hardly give any evidence of the truth of the assertion that these bruits are frequent, for most of you have by this time had a fair experience in the wards and out-patient room, and you know that the *bête noire* of diagnosis, or at any rate one as regards bruits, is the systolic murmur which is heard at the base of the heart, on the left side, over rather a diffuse area. It is so frequent and so difficult to speak of positively. Is it aortic? is it mitral? is it hæmic? are the three more common alternatives, although no one is mutually exclusive of the others. The poor pulmonary artery, however, has from time immemorial been dismissed as not worth a thought, because it is taught to be so rarely diseased, and thus it hardly comes into mind, except in some confused way, as possibly being concerned in hæmic murmurs. Here, as elsewhere, the ingenuity of the vast mass of mind which is everywhere, as Graves says, at work under the stress of the struggle for existence, has acted detrimentally, and the foam of opinion has obscured the solidity of facts.

The latest explanation has had a long run, and well illustrates the axiom that nothing succeeds like success. A distinguished German authority, Professor Naunyn, feeling, I suppose, how unsatisfactory was the explanation that these bruits were hæmic or due to changes in the blood, yet recognising also that they were often, indeed mostly, transitory, and could therefore hardly be due to organic disease; recognising also that in many of them there was a well-

marked systolic apex-bruit, suggested that under varied conditions, chiefly those of anæmia, the left ventricle failed to contract properly, which is true; that a certain amount of regurgitation takes place into the auricle, which is also probably true; and that the murmur is then propagated by means of the auricular appendix, and thus conducted to the surface, which is not true, or at least I do not think so. To support this view it was further urged that the bruit so often present is not of greatest intensity over the pulmonary valves, but in the second left interspace one and a half inches to the left of the sternum, therefore more external than the pulmonary valves. Dr. Balfour, of Edinburgh, has adopted this theory, and, as he thinks, further proved its truth by obtaining cardiographic tracings of the pulse of the said auricular appendix from the chest wall, where the bruit is loudest in intensity. There can, I think, be no doubt that in states of anæmia the left ventricle is often temporarily dilated, or acts badly, and allows of mitral regurgitation; but the bruits of mitral regurgitation are not characterised by their tendency to transmission in the direction indicated. As you know, they ought to be transmitted to the angle of the scapula, and are perhaps also often quite localised to the apex. But mitral regurgitation is, of course, a natural and frequent cause of pulmonary congestion and stasis, and therefore will readily explain some dilatation of the pulmonary artery. To be candid, however, I am not sure that the regurgitation which takes place in anæmia is sufficient to produce such a result, and I only offer the explanation as one which seems at any rate possible, and as having a direct *raison d'être*, whereas the auricular appendix theory has none. I say it has none because, 1st, there is no evidence whatever that the auricular appendix is dilated in cases of anæmia; 2nd, frequent examination in the post-mortem room has made it clear to me that under no circumstances, not even those of its most extreme dilatation, in cases of contraction of the mitral valve, does the auricular appendix come anywhere near the surface of the chest; it is hidden deep back behind the left lung and the right side of the heart. And further, whenever pulsation is felt in this region, it will always be found

to be due, if the opportunity is afforded you of examining it, to a dilated conus arteriosus or dilated pulmonary artery. And I have no doubt, therefore, that the tracings supposed to be *auricular* were in reality derived from the source I have mentioned. The fact is, that whenever there is much mitral regurgitation there is more or less stasis in the lung, and the pulmonary artery and right side become distended, and any distension of the *right* side almost inevitably displaces the left auricular appendix backwards more deeply into the chest. I am of course not now considering such things as aneurism in producing pulsations, or it is just possible that some solidification of the left lung might so alter the relation of parts as to disturb the relative position of the left auricle and allow of its pulsation being conducted more readily to the surface, but I do not remember to have seen such a case.

Few of us, I think, fully realise, unless we have gone into the matter, how deep the left auricle lies. My impression is that we are prone to think of the heart as lying flat, the ventricles as *surfaces forwards* and *backwards;* and I believe this gives rise to a very common error, which is of some practical importance, and to which I may draw your attention in this way. I once went to see a man who was supposed to be suffering from failure of the *muscular tissue* of his heart. He was past sixty, and had rather rapidly failed with shortness of breath, and there being no bruit his first sound had been subjected to careful scrutiny, and had been found, as it was thought, wanting in *power*. His urine, although it contained albumen, was of good specific gravity, and full of urates—admittedly, in fact, a heart urine. I was told, however, that he had had gout several times. He was then suffering from rapidly recurring paroxysms of severe asthma or air hunger, such as one sees in the late stages of renal disease, and when I put my hand upon his pulse I felt a very thick artery and a hardish pulse. I then became rather critically inclined, and on listening to the heart the sounds to my ear were thick and soft, but not wanting in power; indeed, they seemed to me of rather a labouring character, and, in short, I had no doubt that the patient was suffering from Bright's dis-

ease. It is important for my point to add that after the most careful examination I could feel no impulse anywhere. When we got downstairs I expressed my opinion that the case was essentially *renal*, and that the heart although *failing* was nevertheless a *big* heart, and not the feeble, fatty, senile thing that had been imagined. Now, I was not discussing the case with a fool; on the contrary, he was a clever man, and when I told him in effect that he could not diagnose an hypertrophied heart you will not be surprised to hear that he did not much like it. "It seems to me," he said, "that you diagnose it in the absence of all the symptoms that are given in the text-books," and I am obliged to admit the soft impeachment. Yes, I said, I teach at Guy's Hospital that the cases that are usually diagnosed as such are cases of *hypertrophy* and *dilatation*, the latter being in excess, and that the cases of simple hypertrophy, and this even to an extreme degree, may pass unrecognisable so far as the increase of dulness and displacement of the impulse will reveal it. I have verified this over and over again in the post-mortem room. Now why is this? The explanation that I would offer is as follows:—So long as the heart adds to its size in natural model, that is to say, does not change its shape, so long the addition in size is not a material one so far as the *surface* of the *chest* is concerned, for the heart still retains its spiral form, and the left ventricle remains buried, as it should be, under the lung. And this retention of its natural shape is essentially the feature of the enlargement usually present in granular kidney. Of course you often see the other condition in Bright's disease as well, but whenever you see the impulse displaced and the precordial area enlarged you may be pretty sure that something more than simple hypertrophy exists, and this is an important matter for forecasting the issues of the case. And while I am on this matter of displacement of the impulse I will mention another matter which is also worth attention, and which will bring us back to the original question. I have just said that if you find the impulse displaced, &c., you may be sure that something more than simple hypertrophy exists, implying that you may assume that there is both that and dilatation of the left ventricle. But

you must not *at once* assume anything of the sort, although I think you might quite properly do so for anything you are told in books. Before that conclusion is arrived at you must always consider the question whether the displacement may not be due to enlargement or over-distension of the right side. I have drawn on the outline of the thorax the position of the heart as it is in the natural state. It is a copy of a drawing made by the late Dr. Sibson, who devoted much attention to the consideration of the so-called medical anatomy. This drawing accurately represents to my mind the relation of the right side to the surface of the chest. It bulges well over the left, and therefore the more it dilates—that is, the more the imperfection of the working of the left side, for imperfect *left* side action leads to pulmonary obstruction—the less chance there is of any bruit or impulse produced in or by the left ventricle being conducted to the surface. But more than this, I believe many fail to recognise how much the impulse is displaced by what is purely right side disease. I get the impression from the text-books that the right and left side are somewhat separate in their physical action, and that in hypertrophy and dilatation of the right side the cardiac dulness extends to the right, whereas the same condition on the left extends the impulse to the left. To some extent this is so; nevertheless do not forget that displacement of the impulse towards the left and outside the nipple is no uncommon result of hypertrophy and dilatation of the *right* side, and I will add that it is no uncommon case for disease of the right side to be considered to be due to disease on the left, because this point is either unknown or forgotten. It is obvious that the heart not being pinned against the spine, but suspended, tends, whenever it is enlarged, more towards the left.

And now to return to the pulmonary artery, and to the theory that bruits are produced by its dilatation. I think that if you carry this idea into the wards, not as a curiosity of medicine, but as an ever-present *possibility*, you will not have long to wait for some case that shall be confirmatory of it. Here is such a one which will do as well as any. Some time ago I saw a woman who had been recently con-

fined, who, a few days before I saw her, had been somewhat suddenly seized with shortness of breath which was rapidly growing worse. I found her with the right chest dull in front, with a short, dry, pleuritic cough and much dyspnœa; the pulse 120; the heart's sounds, except that they were hurried, were not abnormal. Particular attention was paid to them because it was thought that the case was probably one of pulmonary embolism. She got gradually worse and the chest filled with fluid, and now there appeared a short systolic bruit over the pulmonary artery, accompanied by a slight dry friction-sound, which may possibly have been due in this instance to the distended condition of the pulmonary artery. This bruit remained for nearly three weeks, and during this time it was necessary to tap her three times and to drain the chest, after which she speedily got well. I believe the history of this bruit was as follows :— As the fluid increased in quantity in the right chest, and the breathing became more harassed, so the pulmonary artery became dilated and a bruit appeared; and that when the chest was tapped and drained and the pulmonary circulation relieved, the dilatation subsided, and the symptoms disappeared. I could give other similar cases, and I suspect that it is anything but uncommon in pleuritic effusion.

A man of thirty-seven years was admitted into Clinical Ward on February 5th, 1890. He had had rheumatic fever seven and two years before, but he said that he had enjoyed good health till some ten weeks before his admission, then he began to have a cough and shortness of breath. He had a livid aspect, jaundiced conjunctivæ, slight œdema of the legs, and constant retching, while many of his joints were distorted by old rheumatism.

The heart-sounds are thus described :—Cardiac impulse very much diffused with epigastric pulsation; dulness begins over the fourth left rib, extends on the right to the mid-sternal line, and on the left to the apex, which is in the sixth left space, two inches vertically below the nipple, and one and a half external to this line.

In the aortic area the first sound is rather faint, the second slapping in character, and more accentuated at the mid-sternum opposite the third rib. The first sound is replaced

by a harsh, blowing, systolic bruit as the apex is approached, and is heard on the left of the sternum, and is traceable into the axilla and into the back. The second sound is very faint at the apex, and sometimes cannot be heard at all. There is no diastolic bruit. In the pulmonary area the second sound is also much accentuated, but there is no bruit. In the tricuspid area the sounds have the same character as in the aortic area, but no tricuspid bruit can be heard. There is marked pulsation in the veins of the neck.

He improved very much after a short rest and medicine, and then there appeared for the first time a well-marked diastolic bruit down the sternum, and without thinking much about it I expressed the opinion that in conjunction with the accentuated second sound it indicated some thickening of the aortic valves and regurgitation through them. He left the hospital much relieved, and I had forgotten all about him when he was readmitted under my care into Philip Ward. He was again very ill, and for some time there was no diastolic bruit, but after a time he got better under digitalis, and then the diastolic bruit reappeared, and this happened more than once. I ought to say that the bruit was never very easy to hear, but this was due, I believe, to the extreme accentuation of the second sound which was at all times a marked feature of the case, and which monopolised so much of the attention of our hearing centres that there was hardly time to perceive both it and the bruit within the limits prescribed by the latter. Moreover it was a very local bruit, heard only at the mid-sternum about the level of the third rib, and a little to the left of the breast-bone at the same level.

This disappearance of the bruit and its reappearance, generally, as I thought, as the heart's action recovered itself, set me thinking, and I came to the conclusion that the diastolic bruit was in all probability in this case not aortic, but pulmonary, for the following reasons :—Aortic bruits of diastolic time seldom disappear, for the simple reason that the valvular lesions which produce them cannot alter much. They may, however, sometimes become inaudible for a time, but if this were aortic I never remember before to have noticed one disappear several times and return again.

Then there was the fact that the bruit returned as the heart recovered itself. This is not like the behaviour of an aortic regurgitant bruit, and it is a reasonable suggestion that it was pulmonary in origin, and that it only appeared as the heart's action became stronger, and thus increased the tension in the pulmonary artery. And, as bearing upon this, I have to say, lastly, that the man's symptoms were all pulmonary; he was livid and short-breathed, had a good deal of pulmonary obstruction, and he was on one occasion relieved by leeches applied to the præcordia.

There is one other morbid condition, and another hypothesis based upon it, which has been held to explain the production of a systolic bruit in this particular region. The auricular appendix theory will not bear a critical examination, and failing that, it seems to be admitted on all hands that the functional or hæmic bruits in this particular position are produced in the pulmonary artery. But neither does this theory give any credit to the possible dilatation of the pulmonary artery. Dr. Russell, of Edinburgh, a most acute and close thinker, has put forward the suggestion that the bruit is due to pressure upon the pulmonary artery by a dilated left auricle. But although I agree with Dr. Russell in his demolition of the one theory, I cannot think his own substitute is much more tenable. For although it is allowed that there is often mitral regurgitation in these cases, it cannot be supposed that in the early stages of chlorosis, for example, there is so much regurgitation as to lead to any such dilatation of the auricle as could have any appreciable effect of constriction upon the pulmonary artery. And further, this bruit is often absent, indeed mostly so, in conditions which are far more likely to produce such cases—for example, the common ones of mitral constriction and mitral regurgitation. I have my doubts with Dr. Balfour whether a dilated left auricle *is* capable of producing any appreciable effect by pressure on the pulmonary artery, although in extreme conditions of mitral constriction it is a reasonable hypothesis. But even so, it seems to me that in all these cases the conditions are equally capable of producing, and can be shown to produce in many cases, dilatation of the pulmonary artery, and nothing further is required to explain the presence of the

bruit than it. Further, dilatation of the pulmonary artery has been found in several cases of idiopathic anæmia. Dr. Balfour states that the pulsation in the second left intercostal space is sometimes so well marked and forcible that the late Dr. Hughes Bennett sent him on one occasion a case of chlorosis as a case of aortic aneurism. Its presence under other circumstances has been known to be attended with the production of a bruit; and as a temporary or permanent condition, it is the one I would have you think most of as explaining a clinical phenomenon that is very often met with.

It has been, and it may be contended as an objection to the hypothesis that I am putting forward, that just as I have argued against other theories, so here, if this be so, the bruit is often absent in just such circumstances, so far as stasis of the blood in the lungs is concerned, as it should be most present. But there is an easy reply to that objection, viz. that dilatation of the pulmonary artery is by no means always present when the conditions might seem to favour it. I am afraid that I cannot give you much available help in that matter, but I will say this as worth your thinking over, that you will have in the wards to be aware of and constantly attempting to gauge the relative progression of hypertrophy and dilatation. There is no doubt that under what *seem* very similar circumstances, in one case of heart disease there may be an enormous liver, in another a small one; in one a large spleen, in another a small one; in one a much-congested stomach, in another not; in one hypertrophy in excess of dilatation of the one ventricle or the other, in another the reverse; in one—to come to our present case—much thickening of an artery, in another the hypertrophy may be little and the dilatation enormous. And it may make, often does make, all the difference to the patient. Here, again, the results are often strangely contradictory. It is impossible, indeed, as I have said elsewhere, to mete out in many cases the relative share each takes in the production of the stasis that results, but it is quite clear that the relative extent of the two cannot be unimportant. Extensive dilatation of the right heart, while generally otherwise, certainly allows of the prolongation of life in some cases; extreme hypertrophy, on the other hand, while

on the whole conservative, certainly brings some lives to an abrupt ending.

I will next dwell a little on the cognate subject of the probability of the generation of bruits in simple dilatation of the aorta. I have already spoken of it, but it occurs to me to say that some of the systolic bruits in the aorta have been explained by some to be produced by the blood rushing over the roughened surfaces of the coats of the vessel. But this is at best but doubtful, and an hypothesis of this kind is not necessary if simple dilatation is sufficient for the occurrence in question. Well, then, I beg you to bear the *frequency of dilatation* of the aorta in mind in this respect, because the one idea of many of you is, that if you hear a systolic aortic bruit, it means stenosis of the aortic valves. I am constantly told that there is stenosis of the aortic valves because there is a systolic aortic bruit. But it is generally nothing of the kind: any real stenosis of these valves is one of the most uncommon of valvular affections. I do not suppose that you will see more than two or three such in the course of a year; while a simple systolic bruit over the aortic valves is so common an occurrence that it excites but little interest or thought. Now, what with dilatation of the aorta on the one hand, and obstruction to the smooth passage of blood along it from the valves fringed with vegetations—which is not equivalent to aortic stenosis, mind—on the other, the conditions for the production of a bruit are present in so large a number of cases as to make some cause of this kind a matter of reasonable probability. I will also ask you to remember that the abdominal aorta yields temporarily under conditions of anæmia, and in weak, flabby, neurotic women quite commonly so, and thus gives rise to an abdominal pulsation which to the uninitiated portends the existence of aneurism, whereas it is really of a very much less harmful nature. And with this fact you may also couple another—the common occurrence of not only pulmonary, but also of aortic bruits, at the base of the heart in cases of anæmia. It is no illogical conclusion, I think, that some of these may be due to a temporary yielding of the aortic arch under conditions of mal-nutrition similar to those in the abdominal aorta, and that in both

cases the fixed calibre of the channel is interrupted, and thus a bruit is produced. And from this perhaps you can extract my answer to a question very often asked in the wards: Is a certain bruit aortic or pulmonary? I hold that it is too much the custom to take bruits and treat them generically, so to speak—that is, to assume that they must be either all of one sort or all of the other in cases of anæmia, or the functional or hæmic bruit. I do not think so; I should say that there was as good evidence in favour of the production of murmurs in a simple dilatation of the aorta, in the varied conditions in which functional bruits occur, as there is in favour of pulmonary murmurs; and that in some cases you will find one, in some the other. In many probably both are present, and the place of production of the bruit must be determined by the position of the *greatest intensity*—as far as possible. The presence of this or that bruit is a complex matter which cannot be very well discussed in the abstract; because, as some of you may well be thinking, in some cases of anæmia the aorta is a very small one, and under such circumstances the aortic bruit might well be absent. This, however, is probably only occasional, and then would not apply to relative size; and, too, in such cases the heart is small, the whole cardio-vascular system shrinking together.

But then, perhaps, some one will think that we started on this disquisition upon systolic bruits at the base of the heart upon the strength of a case of dilated pulmonary artery in which the bruit was diastolic; and truly I must allow that, as far as one can see, in that particular case the bruit ought to have been systolic, or at any rate double. Clearly, taking the conditions as they exist, the blood from the right ventricle, flowing past the relatively narrow orifice of the pulmonary artery into the dilated trunk vessel, ought to have produced a systolic bruit; while, if the pulmonary valves had become incompetent, owing to the dilatation of the artery, a double bruit might have been expected, and such a bruit did actually occur in one of these cases of dilated pulmonary artery upon the table. But as a matter of fact it was not so in this case; the bruit was diastolic simply, and unless we suppose that the pulmonary valves were incompetent,

owing to the extreme dilatation of the pulmonary artery, and that the diastolic bruit was produced by regurgitation, one is certainly at a loss for a satisfactory explanation. I am, however, disposed to think that the pulmonary obstruction was so great that the pulmonary valves being intact and competent, the artery was always in a state of distension, and thus may have prevented the occurrence of such fluid veins as would have followed the systole of the right ventricle under conditions of less distension; while the diastolic bruit may be explained by the elastic recoil of the artery after the closure of the valves, and by the consequent vibrations of some portion of the blood.

In connection with this subject, and as possibly helping to explain the difficulty, I may mention that some time ago Dr. Finlayson published two cases of aortic diastolic murmur which occurred in greatly dilated, rigid, atheromatous aortas, but in which there was no disease of the valves. In the first case, one of renal disease, a loud double murmur was heard all over the sternum, and it was naturally concluded that there was aortic regurgitation. But the patient died, and then it was found that, instead of what had been supposed, the state of things that I have described existed. In the second case, again, a loud booming bruit of diastolic time was audible down the sternum, the pulse giving no evidence of aortic regurgitation. The patient died, and the aortic valves were found quite competent, but the aorta was much dilated, highly atheromatous, with calcareous plates and deposit of fibrin. The diastolic bruit is explained by the elastic recoil of the arteries, causing a reflux from the larger vessels back into the dilated and rigid aorta upon the closure of the aortic valves at the onset of diastole.

I have lately seen a similar case: a man sixty years old was sent to me by Mr. Frank Shaw suffering from gall-stones, and from which he died not long after I saw him. He had been known to suffer from aortic disease for a long time, and in accord with that condition he had also suffered from attacks which Mr. Shaw had considered to be due to cardiac failure. I found him with a cardiac impulse far outside the nipple, and with a loud diastolic aortic bruit, which I did not doubt was due to regurgi-

tation through the aortic valves. Unfortunately I have no note of his pulse. At the post-mortem the heart was of natural size, and all the valves were healthy. The aorta was much dilated, atheromatous, and studded with calcareous plates, but the disease did not begin for an inch above the valves. Many of the other arteries were very atheromatous.

This case seems to agree exactly with those recorded by Dr. Finlayson, and the character of the regurgitant bruit was such that it never occurred to me to think that it could possibly be anything else than disease of the aortic valves. It is, of course, possible that the valves leaked, although they gave no evidence of this at the inspection; but this is not at all likely, because the bruit had existed for two years, and no regurgitation of that duration could possibly have been without showing some effect on the valves. One is driven, therefore, to some other explanation, and such a one is found, I think, in some such hypothesis as that given by Dr. Finlayson. But whether that be so or not, we must at any rate recognise the fact that in the great vessels—both aortic and pulmonary—diastolic bruits are occasionally met with which are easily mistaken for those produced by valvular disease.

So much for bruits; but before taking leave of the subject let me say that although fluid veins are capable of explaining many murmurs in the heart as well as in the arteries, nevertheless the question is a much simpler one in the arteries and in the veins than it is in the cavities of the heart. In these latter it is not the mere passage of blood propelled along a simple tube, but blood propelled over a trabeculated and ofttimes highly pouched surface, amid tendinous cords and muscular trabeculæ which may well modify, as well as lead to, the production of sounds. And it is of course owing to the various combinations of circumstances that the varieties of sound are so exceedingly numerous—to wit only for examples the soft bruit de souffle, the rasping bruit of mitral constriction, the several musical bruits, the several diastolic bruits, and so forth. I often think, when cutting open a heart after death, how little idea we *then* get of the pulsatile fulness of its life; of the numberless jets that must be formed as the walls of the

organ pass from the sponge-like condition of diastole into the solidity of the completed systole. One need not wonder at the production of murmurs under circumstances like these; rather should we be surprised that amid such multitudinous although momentary turmoil it is still possible to systematise and discuss certain definite types of cases. To systematise too much, however, would be only to hamper you, for I have found, for my own part, that it is by attempting to realise the physiological conditions of the heart's varied actions one can best interpret the varieties of sound with which we meet; varieties which, although they can be roughly classified according to their time and according to their position, are nevertheless wonderfully different in any one case at one time and at another, and also in separate cases.

A CASE OF GLANDERS,

WITH THE RESULTS OF CULTIVATION AND INOCULATION EXPERIMENTS.

By J. W. WASHBOURN, M.D.

The contagiousness of glanders has been long recognised, but that this is due to the presence of a specific bacillus has only been known for the last eight years.

Babes and Havas ('Orvosi hetilap,' March, 1882) and Roszahegyi ('Pester Med.-Chir. Presse,' 1882, No. 35, case reported by Dirner) described a bacillus found in the pus from human cases suffering from glanders. They, however, made no cultivations of it.

Bouchard, Capitan, and Charrin ('Bullet. de l'Académie de Méd.,' 1882, No. 51, and 1883, No. 41) made cultivations in fluid media from the pus in a human subject suffering from glanders. With these they successfully inoculated horses and donkeys, but from their description it appears that their cultivations were impure.

Löffler and Schütz ('Deutsche med. Wochenschrift,' 1882, No. 52) in a preliminary paper announced the discovery of the bacillus of glanders, giving a brief account of the results of cultivations and inoculations.

Wassilieff ('Deutsche med. Wochenschrift,' 1883, No. 11) described the bacilli in the pus and blood from a patient suffering from glanders.

Israel ('Berliner klin. Wochenschrift,' 1883, No. 11)

shortly after this published the results of cultivations made from the tissues of a horse suffering from glanders. It is doubtful if he was dealing with the same bacillus as that described by Löffler and Schütz, inasmuch as he was able to stain them in sections with watery solutions of methyl violet.

Kitt ('Jahresbericht d. Kgl. Central Thierarzneischule in München,' 1883–4; 'Leipzig Vogel Dist.,' 1884–5) and Weichselbaum ('Wiener med. Wochenschrift,' 1885, Nos. 21, 22, 23, and 24) fully confirmed the observations of Löffler and Schütz. The case described by Weichselbaum was the first case in which the bacilli were cultivated from the human subject.

Löffler ('Arbeiten a. d. Kaiserlichen Ges. Amt.,' Berlin, 1886, Springer) next published his important monograph, in which he gives a full description of the bacillus and the results of cultivation and inoculation experiments.

His observations have been fully confirmed by a number of writers, and the following description is mainly taken from him.

The bacilli are short rods, either straight or slightly curved, and with rounded ends. They are very similar in appearance to tubercle bacilli, but are shorter and thicker. According to Löffler they vary in length between one third and two thirds the diameter of a red blood-corpuscle (2·6—5·5 μ); but according to Weichselbaum very short rods, almost like cocci, may occur.

As a rule the bacilli are single, but two are often found united longitudinally. In old potato cultures threads eight to ten times the length of single bacilli are sometimes found. In liquid media the bacilli are shorter and thicker than those grown on blood-serum. They are non-motile, but possess active Brownian movements. In stained preparations, unstained portions are frequently seen, especially in old potato cultures, and in specimens stained with methylene blue. It is doubtful whether these unstained portions are spores or only vacuoles. The fact that the bacilli quickly lose their virulence when dried is opposed to the theory of spore formation, but it should be mentioned that Löffler in one case found the bacilli virulent after being kept in the dry state

for three months. Baumgarten ('Centralblatt f. Bacteriologie u. Parasitenkunde,' 1888, Bd. iii, No. 13) considers the unstained portions to be spores, inasmuch as Rosenthal was enabled to stain them by means of Neisser's method for staining spores.

In the tissues the bacilli have the same appearance as in the cultures, and often show similar unstained portions. They are found in the nodules, and chiefly near the centre where the cells are breaking down. They mostly lie between the cells, but are sometimes found within the substance of cells which have then generally lost their nuclei. They have rarely been found in the blood, the urine, or the bile (Weichselbaum, loc. cit.; Philipowicz, 'Wiener med. Blätter,' 1885; Ferraresi and Guarnieri, 'Atti della Acad. Med. di Roma,' anno xiii, 1886–7, ser. 2, vol. iii). They have also been found in the tissues of the fœtus when the mother has been affected (Löffler, loc. cit.; Cadéac et Malet, 'Rec. de méd. vétérin.,' 1886; Ferraresi and Guarnieri, loc. cit.).

In cover-glass preparations the bacilli stain with the aniline dyes in watery solutions, but better results are obtained by using alkaline methylene blue (3 c.c. potash solution, 1 in 10,000, and 1 c.c. concentrated alcoholic solution of methylene blue), or with equal parts of Ehrlich's gentian violet or fuchsin and caustic potash solution, 1 in 10,000. The cover-slips should be left in this solution for five minutes, and then placed for one second in 1 per cent. acetic acid which has been rendered sherry-coloured by the addition of trapäolin 00. They should then be washed in water, dried, and mounted in Canada balsam.

In sections the bacilli are difficult to demonstrate. They do not stain at all with watery solutions of the aniline dyes. Löffler recommends the following. Place sections for five to ten minutes in alkaline methylene blue. Decolourise with sulphurous and oxalic acid for five seconds (two drops concentrated sulphurous acid, one drop 5 per cent. oxalic acid, 10 c.c. distilled water). Dehydrate in alcohol, clear in cedar oil, and mount in Canada balsam.

Kühne ('Practical Guide to the Demonstration of Bacteria,' translated by V. D. Harris, 1890) recommends carbolic methy-

lene blue. The sections are placed in this solution (alcohol 10 c.c., methylene blue 1·5 grm., carbolic acid 5 per cent. solution 100 c.c.) for about half an hour. They are then decolourised with acidified water (10 drops of HCl to 500 c.c. of water), and well washed in distilled water. They are now just dipped into absolute alcohol and transferred to methylene blue aniline oil (a small portion of methylene blue dissolved in aniline oil). They are then transferred to pure aniline oil, and cleared in thymin or terebin, and, after washing in xylol, mounted in Canada balsam. He also recommends the dry method, thus doing away with the danger of decolourisation while dehydrating the tissues. The sections are stained in carbolic methylene blue, decolourised in acidified water, washed in distilled water, and floated on two cover-slips. They are then dried on a polished glass plate heated by a spirit-lamp, placed in ethereal oil, and afterwards transferred to xylol and mounted in balsam.

The bacilli will grow on potato, blood-serum, agar, and in broth at temperatures between 22° C. and 42° C. The best temperature is between 37° C. and 38° C. In gelatine growth only takes place at a temperature which produces liquefaction. No growth takes place in infusions of hay, straw, &c.

On blood-serum at 37° C. the growth appears on the third day in the form of yellowish, transparent, tenacious colonies, while at the same time the condensation water becomes turbid. The colonies in eight to ten days assume a milky-white colour, and lose their transparency. This is due to the formation of crystals. On agar, the growth appears in the form of greyish-white drops, which coalesce, forming a layer on the surface.

The addition of glycerine renders the growth more luxuriant (Kranzfeld, 'Centralblatt für Bacteriologie u. Parasitenkunde,' Bd. ii, 1887, No. 10, p. 273).

According to Raskina ('Uratsch.,' 1877, Nos. 40 and 41; reference, 'Zeitschrift für wissenschaftliche Mikroscopie,' Bd. iv, 1887, Heft 4, p. 502), the bacilli grow well at the ordinary temperature of the air on milk-serum-gelatine. In broth at 37° C. a turbidity appears at the end of the second

or beginning of the third day, and afterwards a whitish slimy mass sinks to the bottom.

On the surface of potato kept at 37° C. the growth is very characteristic. On the second day a delicate transparent yellow sticky layer appears, looking like a layer of honey spread on the surface. After six to eight days this loses its transparency, and assumes a reddish hue like that of copper oxide, while around the growth a faint greenish hue appears on the potato. According to Weichselbaum, the colour of the growth is of a dark brown.

With regard to inoculation with the bacilli of glanders, it is found that many animals are susceptible, while others enjoy immunity. The most susceptible are the horse and donkey among the larger animals, and the field-mouse, wood-mouse, hedgehog, and guinea-pig among the smaller animals. Cattle, white mice, and rats enjoy immunity; while rabbits, sheep, dogs, and pigs are only slightly susceptible.

The disease runs a somewhat different course in different animals, but at the same time presents a general similarity.

In the *horse* and *donkey* the disease generally begins in the nose with inflammatory swelling and formation of nodules in the mucous membrane. These ulcerate and give rise to a discharge of thin blood-stained pus. The inflammation and formation of nodules spreads to the neighbouring air-cavities, and ultimately to the larynx and trachea. The corresponding lymphatic glands become affected, and finally nodules appear in the internal organs. The lungs are especially liable to be attacked, and the nodules appear in the form of abscesses having characters intermediate between metastatic abscesses and caseating tubercles.

In some cases (Farcy) the disease begins by the formation of ill-defined nodules in the skin. The lymphatic vessels leading from these nodules become thickened, and form indurated cords, and soon the lymphatic glands become enlarged. The animal may either die of cachexia in this stage, or a general infection may ensue, affecting the nose and internal organs. In slightly susceptible animals, such as the rabbit, the disease may assume a purely local character, ending generally in recovery.

I will describe more fully the disease as it occurs in guinea-

pigs and field-mice after inoculation, inasmuch as these animals are the most suited for inoculation experiments.

In *guinea-pigs* for the first two or three days after inoculation nothing particular is to be noticed. From the third to the fifth day induration occurs at the seat of inoculation, an exudation of pus takes place, and ultimately a scab forms. About the end of the first week the corresponding lymphatic glands become enlarged, and later suppurate and open externally. In some cases the process stops here, the lesions retrogress, and the animal recovers. In the large majority of cases, however, the disease progresses to a fatal termination. The testes and epididymis become swollen and tender, and abscesses form and open externally. In the female a corresponding suppuration takes place in the labia and mammæ. The joints of the foot then suppurate, and nodules appear under the skin, which suppurate and leave ulcers behind. The mucous membrane of the nose only becomes affected in one-third of the cases. When affection of it occurs it shows itself by snuffling breathing and sneezing. Profuse discharge is rare. Death generally occurs very rapidly after the disease appears in the nose. On an average death takes place in three to four weeks, sometimes as early as the end of the first week, and sometimes as late as the end of the eighth week.

Post-mortem examination reveals the following changes: —At the seat of inoculation there is either an indurated swelling or a scar. The corresponding lymphatic glands are swollen, and either show small foci of suppuration or are completely transformed into abscess cavities with thick walls and puriform contents. The joints of the foot may be found suppurating. In the skin small abscesses may be found. The lungs may contain submiliary or larger nodules of greyish-yellow colour, often surrounded by a red zone, or even by well-marked hepatisation. The spleen is almost always affected; it is enlarged, and full of submiliary or larger yellowish nodules, which project above the surface. The liver often contains similar nodules. The kidneys are not affected, but nodules are sometimes found in the suprarenal capsules. The peritoneum and subperitoneal tissue often contain nodules with pus in their interior. The intestinal tract is not

affected. The testes and epididymis at first contain greyish-red nodules of the size of a pin's head, and later larger yellow cheesy foci, breaking down and opening externally. The ovaries rarely contain isolated yellowish nodules. The mammæ frequently contain abscesses. The mucous membrane of the nose is reddened and swollen. The septum and turbinated bones are covered with small lenticular nodules. The bones are sometimes affected, and perforation of the septum may occur.

According to Straus ('Archives de Méd. expérimental,' May 1st, 1889), if inoculation is made into the peritoneal cavity death takes place in from twelve to fifteen days. If the animal is killed as early as two days after inoculation the peritoneum is found healthy, but the tunica vaginalis of the testes is covered with yellowish-white granulations of the size of a pin's head. Later the two layers become united by a thick purulent exudation, and adhesions are formed with the skin. He recommends this method of inoculation as a rapid means of diagnosing a doubtful case of glanders.

Field-mice, when inoculated with the bacilli of glanders, die as a rule in three or four days; sometimes death takes place as early as the first day, and sometimes as late as the eleventh. For the first twenty-four or even forty-eight hours they appear quite unaffected. They then become apathetic, respiration is quickened, and food refused. Suddenly they fall over on the side and are dead. There is never any discharge from the nose. Post-mortem examination reveals a greenish-grey infiltration in the subcutaneous tissue at the site of inoculation. The lymphatic vessels leading from this are swollen, forming indurated cords. The corresponding lymphatic glands are enlarged, and often contain a number of minute whitish deposits. The spleen is much enlarged, and full of minute yellowish-white nodules projecting above the surface. The liver contains excessively minute grey deposits. The kidneys, testes, and mucous membrane of the nose are unaffected. In the suprarenals and ovaries there are sometimes found minute ecchymoses or single yellowish-white nodules. The lungs are usually not affected, but occasionally ecchymoses appear on the surface, and there may be lobular infiltration. The joints

are seldom affected; the skin and subcutaneous tissues never.

Microscopically the nodules in animals are found to consist of epithelioid and small round-cells, and belong to the so-called granulation tumours. The small round-cells rapidly increase and then break down, forming abscesses (Virchow, 'Die krankhafte Geschwülste,' Bd. ii, p. 543; Bollinger, 'v. Ziemssen's Hdb. d. spec. Pathologie u. Therapie,' Bd. iii; Baumgarten, 'Pathologische Mykologie').

In the *human subject*, according to Fagge ('Principles and Practice of Medicine'), the disease may either assume an acute or a chronic form.

In the acute cases the chief symptoms are fever, together with a cutaneous eruption of papules rapidly forming vesicles, and thenceforth pustules which rupture and discharge a thin, purulent, and often blood-stained fluid. There are besides hard painful lumps in the muscles and subcutaneous tissue, which rapidly suppurate. The lymphatic glands are seldom affected. The joints not infrequently suppurate. In some cases the mucous membrane of the nose becomes swollen and ulcerated, giving rise to a thin, purulent, blood-stained discharge. Ulcers may also appear in the mucous membrane of the pharynx and larynx.

In the chronic cases intractable ulcers with thick, livid edges are the most characteristic symptoms. Abscesses around the joints, inflammatory swellings in the skin or muscles, or an eruption of pustules as in the acute form may occur. The nasal mucous membrane may be also similarly affected. In some cases the chief symptoms are cough with sanguineous expectoration.

After death in either case the changes found are often similar to those in pyæmia, but nodules and more or less extensive patches of catarrhal pneumonia which have no pyæmic characters are also seen. Virchow describes the disease in man as consisting of pustules and abscesses, with little to distinguish it from simple inflammation. He, however, says that on careful examination characteristic small yellow bodies may be seen in the mucous membrane of the nose and frontal sinuses, and even in the larynx and trachea.

Abroad, cases of glanders in the human subject from which

the bacilli have been cultivated and successfully inoculated on animals have been reported by Weichselbaum, Löffler, Kranzfeld, Kieman, Ferraresi and Guarnieri, and others. There are at least two cases in which the disease has probably been contracted through accidental inoculation with cultures of the bacillus ('Lancet,' Nov. 2nd, 1889; 'St. Petersburger med. Wochenschrift,' 1890, No. 20). In England, however, the bacilli do not appear to have been cultivated from human cases, and I have consequently considered the following case worth recording.

In this case, of which a full report is given below, the symptoms and post-mortem appearances, although suggestive of glanders, were not conclusive because of the absence of any evidence of infection. It was only by bacteriological investigation that a positive diagnosis was attained. In cover-glass preparations of the pus taken from the subcutaneous abscesses and stained with watery solutions of the aniline dyes, bacilli in large numbers, similar to those described by Löffler and Schütz, were found. They varied in length from 2·5 to 6·25 μ, and were rather thicker than tubercle bacilli; they were either straight or slightly curved, had rounded ends, and showed unstained portions, especially in specimens stained with methylene blue (vide Fig. 1, and compare with Cornil and Babes' 'Les Bactéries,' ii, p. 249). In sections similar bacilli were found both in the walls of the abscesses and in the nodules in the lungs. Here, however, much greater difficulty was experienced in demonstrating the organisms. They would not stain at all in the watery solutions of the aniline dyes, and were decolourised by Gram's method, and by the method for staining tubercle bacilli. The following method, however, showed the bacilli well, but it requires a little experience to get the proper amount of decolourisation. Place the sections in Kühne's carbolic methylene blue for ten to thirty minutes. Wash well in water. Pass rapidly through weak hydrochloric acid solution (Kühne's), and again thoroughly wash in water. Dry the section well on a slide, either over a flame or in the incubator, and mount directly in Canada balsam dissolved in xylol. Both the bacilli and the nuclei are stained blue, but the nuclei are more faintly coloured, and thus do not obscure the darker stained bacilli.

In the walls of the abscesses a large number of bacilli could thus be made out, more particularly towards the inner surface where the cells were breaking down. As a rule the bacilli were single, but sometimes little groups of two or three were found. Most of the bacilli were situated between the cells, but it was not uncommon to find them within the cells, which had then gradually lost their nuclei. In the nodules in the lungs only a small number of bacilli could be found among the small-celled infiltration.

The presence of these bacilli was almost sufficient to diagnose the disease, but it was thought advisable to confirm this conclusion by making cultivations and inoculations. Two rabbits (see p. 141) were inoculated in the subcutaneous tissue of the back with the pus taken from an abscess after death. Local induration occurred, but this subsided, and no general infection followed. This is quite in accordance with the view that the case was one of glanders.

Potatoes were inoculated with the pus taken from an abscess during life. They were placed in damp chambers, and kept in an incubator at a temperature of 30°—37° C. On the second day growth appeared in the shape of a moist yellowish sticky layer, looking like a layer of honey spread on the surface. This growth was found to consist of bacilli similar to those described below. A guinea-pig (A) (see p. 142) was inoculated in the subcutaneous tissue of the back with a minute quantity of this growth. Unfortunately the potatoes became grown over with moulds before the later more characteristic colour was developed.

An agar tube was inoculated with the same pus taken during life, but only an impure culture was obtained. A guinea-pig (B) (see p. 143) was inoculated in the subcutaneous tissue of the back with a minute quantity of this growth.

Both guinea-pigs A and B died in about three weeks from typical glanders. Induration occurred at the seat of inoculation, the neighbouring lymphatic glands became enlarged and suppurated, abscesses formed in the testes or around the vulva, ulcers appeared over the body, and the spleen was found after death to contain numerous minute grey nodules. Bacilli in every respect similar to those in the human case were found in the affected tissues (vide Fig. 2).

Tubes containing agar, broth, potato, and gelatine were inoculated with pus, the juice of the spleen, and blood from the heart, all being taken from the guinea-pigs. No growth took place in any of the tubes inoculated with blood, nor in any of the gelatine tubes, which latter were kept at the temperature of the laboratory. In the other tubes, however, growth occurred.

On the potatoes, which were kept at 30°—37° C., a thin transparent yellowish sticky layer appeared on the second day. This growth gradually lost its transparency, and assumed a characteristic rich reddish-brown colour (vide Fig. 5). There was no greenish hue round the growth as described by Löffler, but this was probably due to the fact that the potatoes were in small slices kept in a test-tube, and not large portions in a damp chamber. Microscopically the growth consisted of non-motile bacilli, possessing very active Brownian movements. They were either straight or slightly curved, and possessed rounded ends. They stained, though with difficulty, in watery solutions of the aniline dyes, and sometimes showed well-marked unstained portions. They were either single, or two or more were united longitudinally. They were shorter than those found in the tissues, and varied in length from 1·8 to 3 μ (vide Fig. 3). Sometimes quite short rods, such as those found by Weichselbaum, were found. In the agar tubes kept at 30°—37° C. growth appeared on the second or third day in the form of whitish colonies, which soon coalesced and formed a layer on the surface; a turbidity also appeared in the condensation water. Microscopically the growth consisted of similar bacilli to those on the potatoes.

The growth in the broth tubes kept at 30°—37° C. appeared on the second day as a turbidity, which ultimately formed a sticky layer at the bottom of the tube. Microscopically the bacilli generally showed well-marked involution forms. They were as a rule 2·5 μ long, but some were as long as 7·5 μ, and they were thickened irregularly so as to assume all kinds of fantastic shapes (vide Fig. 4).

All the growths rapidly lost their virulence, so that they no longer grew when transplanted. This must partly be attributed to the variability of the temperature due to temporary difficulties with the gas supply in the laboratory.

Two guinea-pigs (C and D) (see pp. 145 and 146) were inoculated with cultures obtained from guinea-pigs A and B. They both developed typical glanders, and the bacilli were demonstrated in the tissues after death. Cultivations made from the pus of one of these showed similar growths to those made from A and B. No growth took place on gelatine kept at the laboratory temperature, nor was there any growth in the tubes inoculated with the blood. The bacilli rapidly lost their virulence, and were no longer capable of transplantation, and two guinea-pigs inoculated some weeks afterwards were not affected.

There was now no doubt about the nature of the disease. The presence of the bacilli in the tissues, the characters of the growth, and the results of inoculation were quite conclusive.

The following clinical history of the case is taken from notes by Mr. Clowes.

H. A—, æt. 53, an engineer, living in Bermondsey, was admitted into Guy's Hospital under Dr. Pavy on November 4th, 1889, at 2 p.m., complaining of pains in the limbs and general malaise.

Previous history.—Has been abroad, but not for the last seven or eight years. Works with machinery, and has had nothing to do with horses. Has suffered from cough for the last three winters, but has otherwise enjoyed good health. For the last three years has been a heavy drinker.

History of present complaint.—Says he caught cold three weeks ago, and was obliged to take to his bed. He suffered from pains in the head and limbs and increasing weakness. Four days ago he attended the hospital, but was not then considered sufficiently ill to be admitted. Since this time he has become worse, and painful lumps have appeared over his body. The first to appear was situated behind and below the left ear, the next on the right side of the neck, while to-day a large one appeared over the left scapula. For the last two nights he has been delirious, and has passed his urine under him.

Condition on admission.—Patient appears very ill and weak, and his mental condition is confused. He complains of pain and tenderness in the limbs and back. Temp. 103·4°,

pulse 100, resp. 20. The tongue is dry, and the lips covered with sordes. The breath is foul. The cardiac sounds are feeble; no bruit. There is cough, with slight expectoration of blood-stained mucus. A few rhonchi to be heard over right lung. Urine dark coloured but clear, sp. gr. 1030; no albumen. Below and behind the left ear there is an ulcer with yellow sloughy base, surrounded by much induration. A similar but smaller ulcer is situated on the right side of the neck. Over the left scapula there is an eruption of large pustules. Smaller pustules are present on the shoulders and legs.

Was put on beef-tea, eggs, and milk, and ordered diffusible stimulants.

In the evening patient was delirious. Rusty expectoration; dulness, and fine râles at left base. Several lumps appeared over the body and limbs, but chiefly on the arms. The smaller ones were like pimples, and many rapidly became pustular; the larger ones were at first indurated, but soon became soft and fluctuating.

November 5th.—Temp. 104·4°, pulse 108, resp. 34. Dulness over left base increased. Pleuritic rub audible. Several fresh lumps have appeared on the face. There is a pustule on the right eyelid, closing the eye, and there is slight discharge from the eye. Ordered quinine. An abscess in the arm was opened at 11 p.m., and pus removed for bacteriological examination.

6th.—Worse. Has not slept. Fresh lumps have appeared. Temp. 104·4°. Rhonchi over front of right lung. Back not examined, as patient is too ill. At 4.30 p.m. patient stopped breathing, and almost immediately afterwards the heart ceased to beat.

A post-mortem examination was made on November 7th by Dr. Shaw, and I am indebted to his notes for the following:

Body well nourished. No œdema. On the face, neck, and limbs there are, roughly speaking, four kinds of lesions.

1. Fluctuating swellings about the size of walnuts, with the skin over them unaffected.

2. Smaller, harder lumps, with the skin over them discoloured. Of these there are only three or four.

3. Pustules. The larger ones consist of aggregations of smaller ones.

4. Superficial ulcers with thickening around them. Of these there are only three well-marked examples,—one behind the left ear (which patient indicated as the earliest symptom), a second on the nape of the neck, and a third on the upper eyelid.

On making incisions into these lesions their different appearance was found to be due simply to the position of the suppuration. A typical example of (1) showed a superficial mass of gelatinous pus between the deep fascia and the muscle, and close beside this was another abscess situated within the substance of the muscle, giving rise to no external manifestation. In (2) the suppuration was going on in the deep parts of the skin. In the case of the pustules the suppuration was superficial, and the ulcers were evidently a later stage of these.

None of the lymphatic glands were enlarged. Many joints were opened, but no pus was found. The brain and cord were not examined. The cornea and conjunctiva were not affected. The cavities of the mouth, nose, and pharynx were carefully examined, but no sign of ulceration nor inflammation could be detected.

The heart weighed 9 oz., appeared quite healthy, and contained a well-formed clot.

The liver weighed 59 oz., and was a trifle fatty. The spleen weighed 4 oz., was rather soft, but otherwise healthy. The kidneys together weighed $10\frac{1}{2}$ oz., and were healthy. The testes, stomach, and intestines were healthy.

The apices of both lungs were adherent from old pleurisy. Some phthisis, both old and recent, was present at the left apex. The pleuræ over both bases were acutely inflamed, the right more than the left. There was considerable suppuration in both lungs, and the pus in the abscesses had the same semi-consistent gelatinous appearance as in the subcutaneous abscesses. There was a zone of pneumonic consolidation around the abscesses. There was also a patchy broncho-pneumonia unconnected with the abscesses. The distribution of the suppuration was curiously unlike in the two lungs. In the left lung it was limited to the lower lobe, and was very diffusely scattered, and a great many abscesses were visible as yellow projections beneath the pleura. In

the right lung nothing was to be seen from outside, but on cutting into the lung a large number of abscesses were found collected around the root.

The larynx, trachea, and bronchi were extremely red, but this was considered by Dr. Shaw to be rather due to post-mortem staining than to inflammation.

On microscopical examination the nodules in the subcutaneous tissue and skin were found to consist of an infiltration of small round-cells which were breaking down to form abscess cavities. Many bacilli were found. In the lungs there was found an infiltration of small round-cells, also breaking down to form abscess cavities. The alveoli around were full of blood. Here only a few bacilli could be demonstrated. It will be remembered that the pus removed during life from the abscess on November 5th showed numerous bacilli. Tubes containing potatoes, agar, and gelatine were inoculated with this pus. Growth, as already described, occurred on the potato and agar.

On November 6th, two hours after death, another abscess in the arm was opened. Cover-slip preparations showed similar bacilli to those found in the pus removed during life.

The following are the details of the results of the inoculations.

Result of Inoculation.—Two Rabbits inoculated on November 6th.

Two rabbits (already mentioned, p. 136) were inoculated in the subcutaneous tissue of the back with the pus from the abscess opened after death.

Two days later some induration appeared at the seat of inoculation in both rabbits, and a crust formed. This induration increased, and was well marked on November 11th. After this date, however, it gradually decreased, and by November 25th had quite disappeared. One rabbit was killed on January 29th, and all the organs were found perfectly healthy. Nothing was to be seen at the seat of inoculation. The other rabbit began to take food badly on December 11th. This continued, and it died on December 17th with much emaciation.

Post-mortem.—A superficial scar was found at the seat of inoculation. The liver was full of whitish irregular masses, many being calcareous.

Microscopically these changes were found to be caused by the presence of a number of psorosperms. All the other organs were healthy, and no bacilli could be found in any of the tissues.

Result of Inoculation. — Guinea-pig A inoculated on November 8th.

On November 8th a guinea-pig (A) (already mentioned, p. 136) was inoculated in the subcutaneous tissue of the back with a small portion of the growth from one of the potatoes.

9th.—Slight induration at seat of inoculation.

11th.—Ulcer with dry, raw surface formed at seat of inoculation.

12th.—Skin around seat of inoculation firmly fixed to bone by indurated tissue.

14th.—Induration increased, and spot tender. Slight secretion of pus. Microscopically the pus was found to contain diplococci, short thick bacilli, and a few bacilli similar to those found in the human case.

25th.—A shallow ulcer with dry base about a quarter of an inch in diameter has formed on the abdominal wall. The lymphatic glands in the groins are enlarged and tender, and a tender swelling has appeared around the vulva.

28th.—Animal died in the night.

Post-mortem examination.—An abscess containing about half a drachm of curdy pus was found at the side of the vulva ; the walls of the abscess were adherent to the adjacent bones. The lymphatic glands in the groin were enlarged and suppurating. An ulcer was found on the abdominal wall with some thickening around it. There was an ulcer at the seat of inoculation, with surrounding induration binding it down to the bone. Healthy tissue intervened between the seat of inoculation, the ulcer, the abscess cavity, and the glands. The spleen was dotted over with minute whitish nodules about the size of a pin's head, many of which pro-

jected above the surface. The other organs were healthy. No ulceration was found in the nasal mucous membrane.

Microscopically the following appearances were found.

The *spleen* did not present very marked changes, but foci were found where the nuclei appeared shrivelled and granular, and either stained badly or not at all with logwood.

The *lymphatic glands* showed an increase of small round-cells, which were breaking down to form abscess cavities. Many bacilli were demonstrated in the walls of the abscesses.

In the *liver* were small scattered foci, consisting of small round-cells, with several nuclei staining well with logwood. No bacilli could be found.

The *kidneys* were normal, and no bacilli were found.

The walls of the *abscess* around the vulva were infiltrated with small round-cells, some containing several nuclei. Many bacilli were found.

At the *seat of inoculation* there was a similar infiltration of small round-cells. Many putrefactive bacilli were found on the surface, and in the depths of the tissue a few bacilli similar to those in the glands and the abscess were seen. Inoculations were made on agar, broth, and gelatine with the heart-blood, juice of spleen, and pus from the abscess and gland. The tubes of agar and broth inoculated with the pus and the juice of the spleen showed growths with characters similar to those already described. No growth occurred in the gelatine tubes, nor in any of the tubes inoculated with the blood.

Result of Inoculation. — Guinea-pig B inoculated on November 14th.

On November 14th a guinea-pig (B) (already mentioned, p. 136) was inoculated in the subcutaneous tissue of the back with the impure agar culture from the human case.

15th.—Induration at seat of inoculation.

26th.—Induration has gradually increased. Both testes are swollen and tender, and the inguinal glands are enlarged.

30th.—A shallow ulcer a quarter of an inch in diameter, and with a dry glazed base, has appeared on the left leg.

December 2nd.—Drowsy. Slight inspiratory dyspnœa and slight muco-purulent discharge from nose.

4th.—Marked inspiratory dyspnœa.

5th.—Dead.

Post-mortem examination.—There was an abscess containing cheesy pus in the upper part of each testis and epididymis, which were swollen to size of pigeon's eggs. It appeared to commence in the epididymis, but the testis was also involved. There was a shallow ulcer on the left leg about a quarter of an inch in diameter.

The *lymphatic glands* in the groin were enlarged, and showed small points of suppuration.

There was induration and slight suppuration around the *seat of inoculation.*

The *spleen* was enlarged and full of minute whitish nodules. In the liver were a few small irregular whitish patches.

The other organs were healthy. No changes were seen in the nasal mucous membrane.

The histological changes were similar to those in guinea-pig A.

Bacilli were only demonstrated in the walls of the abscess in the testes and in the foci in the spleen, and these only in small numbers.

Inoculations were made on potato, agar, and broth from the heart-blood, juice of spleen, and pus from glands and testes. Growth appeared with characters already described in the potato, broth, and agar tubes inoculated with the pus and juice of spleen. No growth occurred in tubes inoculated with the blood. The growths were successfully transplanted on potato and agar, but on solid gelatine transplantation was found impossible.

On December 2nd a guinea-pig (C) was inoculated with a small portion of a growth on agar obtained from the spleen of guinea-pig A, and another guinea-pig (D) with a growth on agar obtained from the pus of the same guinea-pig.

Result of Inoculation.—Guinea-pig C inoculated on December 2nd.

December 6th.—Marked induration at seat of inoculation. This gradually increased.

9th.—Enlarged and painful gland in left groin. Painful swelling around vulva.

21st.—Small ulcer in right inguinal region discharging pus. Adjacent to it another ulcer commencing.

26th.—Ulcer no longer discharging. Swelling around vulva spreading to groin. Small ulcer appearing behind left ear. Animal ill.

28th.—Two large tender swellings in both inguinal regions. Ulcer forming over the left sac.

January 2nd.—Dyspnœa.

4th.—Death.

Post-mortem appearances.—Ulcers on either side of vagina, behind left ear, and on middle of abdominal wall. Glands in both groins full of white curdy pus. Abscess around vagina. Induration and scab at seat of inoculation. Peritoneum healthy. Abscess containing curdy pus behind the peritoneum communicating with the seat of inoculation by the side of the vertebræ, which are bare. Nasal mucous membrane healthy. Spleen full of minute whitish nodules. Other organs healthy.

Microscopical examination revealed changes in the liver and spleen similar to those in the other guinea-pigs. No bacilli could be demonstrated. The glands in the groin, the ulcers, and the seat of inoculation showed an infiltration with small round-cells. Bacilli similar to those already described were found in the glands and in the deeper parts of the small-celled growth around the seat of inoculation. Near the surface of the latter and of the ulcers putrefactive bacilli were found.

No cultivations were made from this case.

Result of Inoculation.—Guinea-pig D inoculated on December 2nd.

December 3rd.—Slight induration at seat of inoculation. This gradually increased.

8th.—Testes slightly swollen.

9th.—Tender enlarged glands in both groins.

14th.—Shallow ulcer with dry base on anterior abdominal wall.

19th.—Killed with chloroform.

Post-mortem appearances. — Induration around seat of inoculation. An ulcer close to this with indurated base. A similar ulcer on anterior abdominal wall. Glands in both groins contain curdy pus. Testes enlarged and soft, but otherwise appear healthy. Liver full of minute whitish nodules; two or three small white patches on surface covered with lymph. Other organs healthy.

Microscopical examination revealed changes in the liver, glands, and induration at seat of inoculation similar to those in other guinea-pigs. The spleen and testes showed no changes. Many bacilli were demonstrated in the lymphatic glands of the groin.

Inoculations were made on potato, agar, and broth with blood from the heart, pus from the glands, and the juice of the testis and liver. No growth occurred in any of the tubes inoculated with either the blood, or the juice of the testis and liver.

Growth occurred in the tubes inoculated with the pus, and was similar in every respect to the growths already described.

On January 13th transplantation was attempted, but without result.

On January 9th two guinea-pigs were inoculated, one with a growth from guinea-pig B and the other with a growth from guinea-pig D. Slight induration occurred at the seat of inoculation, but this quickly subsided. Some weeks later the animals were killed with chloroform, and all the organs found healthy.

DESCRIPTION OF PLATE

Illustrating Dr. J. W. Washbourn's Paper on a Case of Glanders.

FIG. 1.—Cover-slip preparation of pus taken during life from abscess in arm. Methylene blue; $\frac{1}{12}$th oil immersion.

FIG. 2.—Section through wall of abscess in lymphatic gland of guinea-pig. Carbolic methylene blue; $\frac{1}{12}$th oil immersion.

FIG. 3.—Cover-slip preparation of bacilli from potato culture. Fuchsin; $\frac{1}{12}$th oil immersion.

FIG. 4.—Cover-slip preparation of bacilli from broth culture, showing involution forms. Fuchsin; $\frac{1}{12}$th oil immersion.

FIG. 5.—Potato culture of bacilli.

The drawings from which this plate was taken were kindly made for me by Mr. G. Sichel.

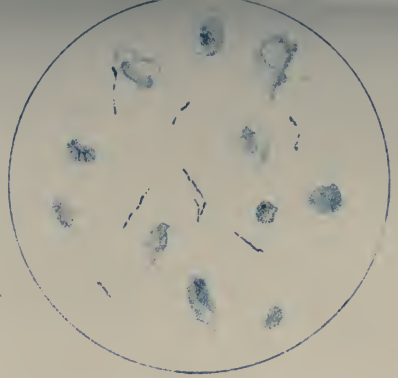

Fig. 1

Fig. 2

Fig. 3

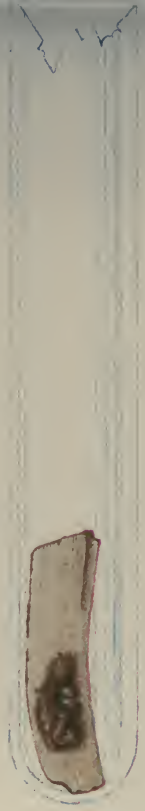

Fig. 5

Fig. 4

G Sichel, del.

Danielsson & Co, chromo

ON THE PATHOLOGY AND PROGNOSIS OF PERNICIOUS ANÆMIA.

By W. HALE WHITE, M.D.

Pernicious anæmia is so closely associated with Guy's Hospital, for Addison[1] first accurately described it, and Drs. Wilks,[2] Taylor,[3] and Pye-Smith[4] have published series of cases in these 'Reports,' that they appeared to me a suitable place in which to give an account of a case of recovery from this disease whose subsequent history I have been able to follow for some years after his restoration to health in Guy's Hospital; for not only are cases of complete recovery very rare, but very few if any have been published in which the recovery has lasted so long.

During the last three or four years some new pathological work has been carried out in connection with pernicious anæmia; the attempt to harmonise the pathological suppositions with the clinical symptoms has brought into prominence certain symptoms. In order to test the accuracy of this recent work, I have given at the end of this paper a short account of each undoubted case of pernicious anæmia which has died in Guy's Hospital since the time of Addison. Many of these cases have already been published. I have always given the reference to the place of publication, but whenever I could find the original report in the hospital records I have read it most carefully in order to search for those symptoms of which so much has been made lately; and

[1] 'On the Constitutional and Local Effects of Disease of the Suprarenal Capsules.'

[2] 'Guy's Hospital Reports,' series 3, vol. iii.

[3] Ibid., series 3, vol. xxiii.

[4] Ibid., series 3, vol. xxvi.

whenever from the brevity of the previously published report they have been omitted, I have inserted them. This paper, therefore, consists of two distinct parts; namely, the prognosis of pernicious anæmia, together with the record of a case of recovery, and the testing of the recent pathological views of this malady by the fatal cases we have had at Guy's Hospital. It will, I think, be easier of arrangement to take the last question first.

Part I.—The Pathology of Pernicious Anæmia.

Before discussing the pathology of pernicious anæmia it will be well to point out what precisely is meant by the term. Addison says, "For a long period I had from time to time met with a very remarkable form of general anæmia, occurring without any discoverable cause whatever—cases in which there had been no previous loss of blood, no exhausting diarrhœa, no chlorosis, no purpura, no renal, splenic, miasmatic, glandular, strumous, or malignant disease. Accordingly, in speaking of this form in clinical lecture, I perhaps with little propriety applied to it the term 'idiopathic' to distinguish it from cases in which there existed more or less evidence of some of the usual causes or concomitants of the anæmic state." This contains the whole pith of our conception of what is meant by the idiopathic anæmia of Addison, or, as it has since been called, progressive or pernicious anæmia. All cases in which the anæmia progresses are not examples of this malady, even if they are fatal, and therefore the term pernicious is perhaps better than progressive, and it is preferred by Fagge. For the case to be an example of the disease described by Addison, it is essential that there should not be present any of the usual causes or concomitants of the anæmic state. Thus malignant disease can never give rise, however severe the anæmia associated with it, to the idiopathic anæmia of Addison, or "progressive or pernicious anæmia." Nor can chlorosis, however severe, ever become "pernicious anæmia;" the "usual concomitants" of the sex, age, and curability by iron separate chlorosis from

it. No doubt it is true that patients who during life have presented the appearance of the idiopathic anæmia of Addison have been found after death to be suffering from malignant disease or phthisis, but this no more proves that idiopathic or "pernicious" anæmia is the same as the anæmia of phthisis or malignant disease than the fact that during life a case of acute intestinal obstruction is mistaken for one of perforated gastric ulcer, proves these two maladies to be one and the same.

Perhaps those who have been trained by Addison or his successors at Guy's may consider these remarks superfluous; but I venture to think they are not, for the idea has got abroad that any anæmia, due to any cause whatever, may, if it be sufficiently prolonged, eventually become the special form of anæmia described by Addison. Thus Henry,[1] speaking of the idiopathic anæmia of Addison which he prefers to call pernicious anæmia, says, "I do not regard it as an independent disease. . . . Pernicious anæmia is the final stage of several forms of sympathetic anæmia and of chlorosis." In an article on anæmia published elsewhere[2] I have criticised this view, and Dr. Henry has replied to my criticism; but I still cannot help thinking that he has failed to grasp precisely the characters of the disease which we at Guy's Hospital call "idiopathic anæmia" or "pernicious anæmia." Anyhow, in the cases which will be described in this paper I shall limit myself exclusively to those in which there have been none "of the usual causes or concomitants of the anæmic state." Of course the word concomitants of the anæmic state does not here mean symptoms such as dyspnœa, œdema of the ankles, hæmic murmurs, hæmorrhages, &c., which are common to all forms of anæmia. The word concomitants is used, with Addison's usual caution, to convey that he is by no means sure that some conditions which appear at first sight to be causes of the anæmia may not be simply concomitant with it, and due together with it to a common cause. For example, it is quite possible that the enlarged lymphatic glands in Hodgkin's disease may not be the cause of the anæmia. Perhaps they are simply concomitant with

[1] 'Anæmia,' G. F. P. Henry, Philadelphia, 1887.

[2] 'Polyclinic,' vol. v, No. 4, Philadelphia, 1887.

it, and both the anæmia and the enlarged glands are due to a common or even different cause of which we are ignorant.

In describing the cases the common symptoms of all forms of anæmia and the well-known symptoms of "pernicious anæmia," such as the lemon colour, which are more or less characteristic, will be omitted for the sake of brevity.

The malady will not be called either "idiopathic" or "progressive," but "pernicious anæmia;" because this term is now more largely used than any other, it being distinctly understood that "pernicious anæmia" does not mean any destructive anæmia, but denotes a special disease.

Another reason for using the term pernicious is that Dr. Stephen Mackenzie[1] has recently suggested the term "idiopathic anæmia" should embrace all primary anæmias; and that it should be a generic term, including several species, one of which is "pernicious anæmia." I myself think that this use of the word idiopathic will lead to confusion, as Addison used it in an entirely different sense.

I will now give a brief abstract in chronological order of the recent papers on pernicious anæmia, and contrast the results at which their authors arrive with the clinical features of the thirty-one cases of the malady which have died at Guy's Hospital since the time of Addison.

All the recent observers to whose cases I shall refer are agreed that the anæmia of pernicious anæmia is a different condition from the anæmia of phthisis, malignant disease, &c., although the latter may very closely resemble genuine pernicious anæmia. In a discussion upon pernicious anæmia, it is even advisable not to treat of that form of anæmia which Fenwick has described as associated with atrophy of the gastric glands, for the two conditions are probably distinct. Also it is agreed that the only change in the blood which is peculiar to pernicious anæmia is that the red corpuscles, although very much diminished in number, contain more than the normal amount of hæmoglobin, so that the decrease in the total amount of this substance is much less than the decrease in the number of corpuscles would lead us to expect. Lastly, pernicious anæmia is unaccompanied by changes in the spleen, the lymphatic glands, and the marrow of the

[1] Lettsomian Lectures on Anæmia, 'Brit. Med. Journ.,' 1891, vol. i, p. 58.

bones. I merely mention these well-known points to show that doubtful cases will not be considered.

Recently much attention has been given to the detection of iron in the liver. The best way of doing this is to place a piece of the fresh organ in a solution of sulphide of ammonium, when, if much iron be present, the tissue quickly turns coal-black; or if the piece of liver be treated with a freshly prepared solution of ferrocyanide of potassium and very dilute hydrochloric acid, a fine blue colour due to prussian blue is developed if much iron be present. The organ treated in this way forms a beautiful microscopical object. The iron which is combined with the hæmoglobin does not give a reaction by either of these methods unless strong hydrochloric acid is used. If a quantitative analysis of the iron be required, it is important that the organ to be analysed should be as far as possible completely washed of its blood and dried.

Many observers have shown that the liver of patients dying from pernicious anæmia contains much more iron than that of those dying of other diseases; and Dr. Hunter,[1] after tabulating a number of cases, and carefully excluding doubtful ones, concludes that while the liver washed of its blood and dried ordinarily contains on the average 0·083 per cent. of iron, that organ washed of its blood and dried contains in pernicious anæmia 0·535 per cent. (cases by Stahel, Zaleski, and Rosenstein), or more than seven times as much. He has found in pernicious anæmia this iron is always in the outer two thirds of the hepatic lobule, and is always most abundant within the liver-cells, while in other diseases it is irregularly scattered throughout the liver. By contrasting the proportion of iron in the spleen and liver both by quantitative analysis and micro-chemical examination, it appears probable that the proportion of iron in the liver to that in the spleen—ordinarily in the dried organs freed from blood there is 0·083 per cent. in the liver, and 0·171 per cent. in the spleen—is greatly increased in pernicious anæmia; the average of two analyses giving 0·566 per cent. for the liver, and in the same two cases the spleen yielded 0·156 per cent.

[1] "An Investigation into the Pathology of Pernicious Anæmia," by Wm. Hunter, M.D., 'Lancet,' Sept. 22nd, 1888.

Thus ordinarily the proportion of liver to spleen is 1 to 2, in pernicious anæmia it is 1 to 0·3—that is to say, there is a great relative increase in the liver. This fact, and, if it should be confirmed, that of the constant position of the iron in the liver in pernicious anæmia, point strongly to the conclusion that in this disease the deposition of iron in the liver is not merely due to decay of the blood-corpuscles and the deposition of their iron—for then probably it would not be found in parts only of the lobule, and it would be deposited as much in the spleen as the liver,—but they indicate that the liver plays an important part in the destruction of the blood in this disease, or at any rate in the disposal of the resulting free pigment. There is a certain amount of evidence that the bone marrow is continually trying to replace the blood-corpuscles destroyed.

So far Dr. Hunter has confined himself to pernicious anæmia in man, and he is confirmed in his observations by the writers whose analyses he quotes, and also by others who have since found a great quantity of iron in the liver. When he comes to the experimental part of his inquiry he is upon much more uncertain ground. We all know how much caution is necessary in interpreting human physiology by the light of the physiology of the lower animals, and still more caution is needed in applying the effects of drugs upon the lower animals to explain a disease which, as far as we know, is limited to man, and presents after death no very marked pathological appearances. While these experiments deserve the most careful consideration, and must be repeated in order to see if they can be confirmed, we must always bear in mind that for them to be conclusive they must succeed in producing in animals that assemblage of symptoms which constitutes pernicious anæmia. However, Dr. Hunter concludes from his experiments that there is present in the body in pernicious anæmia a substance resembling in its action toluylendiamine, which limits its destructive action to the blood in the portal circulation—that consequently iron is stored up in the liver. This poison he thinks is probably of a cadaveric nature; it is produced in the gastro-intestinal tract, and this fact explains the frequent presence of gastro-intestinal symptoms in pernicious anæmia.

In the 'Practitioner'[1] and other periodicals he has published in detail a case of pernicious anæmia. The patient was a man aged fifty-eight, who had all the characteristic symptoms of pernicious anæmia. He suffered much from soreness of the mouth, throat, and tongue. During the last two months of his illness he had diarrhœa. The changes in the blood were those usually present in pernicious anæmia. The red corpuscles were reduced before he died to 19 per cent., and the hæmoglobin 22 per cent. After death the liver showed abundance of free iron.

The urine was very high-coloured, like dark sherry, and it was darkest at a time when the patient was very drowsy, markedly yellow, and passing dark-coloured stools. It never contained bile. Spectroscopically examined, it showed a characteristic absorption band lying close to the F line of the spectrum. The colouring matter which gave this spectrum was separated by shaking the urine with chloroform, evaporating, and dissolving in alcohol, and then also gave an absorption band close to the F line. An alcoholic solution of chloride of zinc added to the urine gave a marked green fluorescence. These facts indicate that the cause of the dark colour of the urine is the presence of pathological urobilin, which has been described by Dr. MacMunn.[2] The presence of pathological urobilin in the urine is possibly a result in some way or another of the destruction of blood which occurs in pernicious anæmia, and the increase of biliary pigments may be due to the same cause. The urine was frequently examined microscopically, and whenever the patient was worse—being more drowsy and of a deeper lemon colour—many degenerate renal cells were seen, containing yellowish pigment granules which resembled blood-pigment very closely in appearance, size, and colour. From this fact, the study of other cases, and other evidence, the conclusion is drawn that ordinarily during pernicious anæmia all the pigment resulting from the destruction of blood in the portal area is arrested in the liver, but when the patient becomes worse it overflows into the general circulation, and is excreted in

[1] 'Practitioner,' vol. xliii, pp. 161, 321, 401; and 'Brit. Med. Journ.,' July 5th and July 12th, 1890, vol. xi, pp. 1 and 81.

[2] MacMunn, 'Journal of Physiology,' vol. x, p. 71.

the urine in such a form that it is not readily detected as iron in the urine, and is selected by the cells of the convoluted tubes—in this respect resembling indigo carmine—not passing out by the glomeruli as does the pigment in paroxysmal hæmoglobinuria. Dr. Hunter founds this statement on the microscopical examination of the kidneys of several persons dying from pernicious anæmia, and he always finds the pigment granules, if any are present at all, in the epithelium of the convoluted tubes. The very few analyses that have been made point to the conclusion that more iron is excreted in the urine in pernicious anæmia than in health. The aromatic compounds in the urine, which are chiefly phenol, indol, skatol, and creasol, are excreted in the urine as aromatic sulphates (B). Sulphuric acid is also present in the urine, combined with the salts of the food (A). In health the proportion of A to B is 10 to 1, irrespective of the food taken ; but if putrefactive changes occur in the intestine the quantity of aromatic sulphates is increased. This was so in this patient ; for although, because of the slight amount of food taken, the quantity of A excreted was slight, B did not diminish equally, so that the proportion of A to B was sometimes 3 to 1 ; but the ratio varied, the amount of aromatic sulphates being greatest when he was worst with drowsiness, contracted pupils, sweating, and was passing high-coloured urine. From this increase in the amount of aromatic sulphates the conclusion is drawn that in pernicious anæmia the putrefactive changes in the intestine are in excess.

It was also noticed that there were in the urine certain ptomaines which are never present in health, one of which was probably putrescine. Now this class of ptomaines is only formed from proteids by bacteria, and is not the result of ordinary putrefactive processes which go on in the intestine ; therefore the conclusion is drawn that these ptomaines are due to some special micro-organism which, as putrescine in itself is not poisonous, produces putrescine as well as other poisons which cause the symptoms of pernicious anæmia. In favour of this view it is urged that the symptoms the patient presented when he was worse—such as drowsiness, perspiration, and contracted pupils—point to

poisoning, and also that at the autopsy it was found, on microscopical examination, that the stomach was the seat of chronic subacute and inflammatory processes, and the lymph-glands in connection with it were swollen.

The conclusion to which this case is taken to point is that in pernicious anæmia there is a special micro-organism in the intestinal tract which gives rise to ptomaines such as putrescine, and to poisons which produce the symptoms of pernicious anæmia. These micro-organisms or their products cause a great destruction of blood in the portal system, which leads to deposit of iron in the liver, to the microscopical alterations in the blood-corpuscles, to the excretion of iron-containing pigment by the cells of the convoluted walls of the kidney, to the increase of iron and the presence of pathological urobilin in the urine, and possibly sometimes to an increased excretion of biliary pigment. The increase of aromatic sulphates in the urine also points to the increased putrefactive changes in the intestine. Certain conditions are particularly favorable to these micro-organisms, such conditions being especially disease of the gastro-intestinal tract. Hence the presence of the gastritis in this case was favorable to these micro-organisms; and this view explains the frequent occurrence of symptoms closely resembling pernicious anæmia in certain gastro-intestinal diseases, such as malignant growths. On this hypothesis of pernicious anæmia, gastro-intestinal symptoms, as diarrhœa and vomiting, are of special importance, for they may be due either to the disease which is favorable to the micro-organisms, or to them, or the products of them.

This view of pernicious anæmia would have much more weight than it at present has if it were founded on more than one case, if such severe gastritis had not been found, and if the report of the case had stated whether the bones were examined, and whether the lungs and large intestine were healthy. No mention is made of these parts, and we all know that disease of each of them is sometimes mistaken for pernicious anæmia. I have had under my care a patient suffering from phthisis who passed large quantities of pathological urobilin.

Mott has recently recorded three cases of pernicious

anæmia. His first[1] was a man aged forty-eight who was admitted into Charing Cross Hospital, March 6th, 1885. His urine was very dark, but there was no diarrhœa till arsenic was administered. As diarrhœa always happened when this was done, it was omitted, and the man was on June 4th sent to a convalescent home, where he tried arsenic again, but had to discontinue it in a few days for the same reason. He got much worse, and was readmitted June 20th with diarrhœa and loss of flesh. The presence of diarrhœa prevented his being treated by arsenic, and he died comatose on July 28th. At the autopsy abundance of iron was demonstrated by the sulphide of ammonium and by the ferrocyanide tests. Quantitative analysis showed twice as much iron in the ash of his liver as in the same amount of liver taken from a man dying of paraplegia.

The second case[2] was that of a woman, aged forty-nine, who did not suffer from vomiting, nor was there any marked intestinal disturbance. The urine was very dark, but both Dr. Mott and Dr. Halliburton were of opinion that although it contained an excess of normal urobilin, there was no pathological urobilin. Analysis of the liver gave the result that 112·3 grammes of liver contained 0·3237 gramme of ferric oxide, whilst in health there should be mere traces. The spleen and kidneys showed a trace of iron.

The third case[3] is that of a boy aged eleven. On admission he suffered from constant retching and vomiting, which it was said had come on during the last day or two. The urine was pale. He suffered from epigastric pain and vomiting; the bowels were relaxed. He died soon after admission.

Autopsy.—The marrow of the middle of the shaft of the femur was red and gelatinous, like red currant jelly. The urine did not give an absorption band at F. The liver and spleen showed abundance of free iron by the sulphide of ammonium and potassium ferrocyanide tests. The liver, which weighed 900 grammes, contained 1·33 grammes of ferric oxide; the spleen, which weighed 100 grammes, contained 0·17 gramme of ferric oxide; and the kidneys and

[1] 'Lancet,' vol. i, 1889, p. 521.
[2] 'Lancet,' vol. i, 1890, p. 287.
[3] 'Practitioner,' August, 1890, p. 81.

pancreas contained traces. The glands of the stomach and intestine were normal.

Dr. Mott considers that one of the normal functions of the liver is to destroy blood and excrete the resulting pigment in the bile; and he looks upon pernicious anæmia as an exaggeration of this process, either due to a disturbance of one of the normal functions of the liver, or to the absorption of some substance from the alimentary canal.

Copeman[1] mentions a case in which there was dark urine which contained an excess of normal urobilin, but there was no pathological urobilin. He[2] has also shown that crystals of hæmoglobin may be very readily obtained from the blood in pernicious anæmia, and he and Bond have shown that the administration of arsenic prevents this.

The important stride we have recently made is, then, that we have discovered there is a great increase of iron in the liver in pernicious anæmia, and that this very probably is due to the fact of increased destruction of hæmoglobin somewhere in the portal system or its annexa. We have thus learnt the probable seat of the disease. We have also learnt that the dark colour of the urine which was pointed out by Dr. Fagge and Dr. Pye-Smith is due to urobilin, although it is not settled whether this be the normal or the pathological variety; and it seems a reasonable hypothesis that this excess of colouring matter in the urine is a result of the increased destruction of blood in the portal system.

We are still in the dark as to the cause of this increased blood destruction. It is very tempting to asssume that it is connected in some way with the intestines, but it is not definitely proved that the liver is not the organ at fault. To settle this we require analyses comparing the portal blood in this disease with that of the general circulation. In favour of the view that the origin of the destruction is in the intestines and stomach, the frequency of diarrhœa, vomiting, and other gastro-intestinal symptoms is urged. We will directly see how frequent they are, but even if they exist in every case this evidence is not conclusive that the primary seat is gastro-

[1] 'Lancet,' vol. i, 1890, p. 375.

[2] 'St. Thomas's Hospital Reports,' 1887.

intestinal, for if the liver is made a storehouse for the products of blood destruction, what is more likely than that abnormal products should be excreted in the bile, and thus they may cause the gastro-intestinal symptoms? Dr. Hunter's hypotheses are drawn largely from a single case, and from experiments on various animals with drugs. Their bases are, therefore, weak, and will require much support before the theory, which is ingenious, can be accepted. He lays much stress on the coincident increase of the symptoms from time to time, and on symptoms which suggest a toxic influence, such as drowsiness, contracted pupils, and sweating. I should, from the cases I have watched, and as a result of reading carefully the reports of thirty-one cases, be inclined to say that these "fits," so to speak, of coincident increase of symptoms are quite the exception in genuine pernicious anæmia, and that toxic symptoms are very rare; and further it must be remembered that drowsiness of itself is of no value as a symptom, for it is often seen in those who are extremely weak from any cause. It has been urged against this theory that gastro-intestinal lesions of all sorts are common, and bacteria are plentiful in the intestine, and that therefore pernicious anæmia should be commoner than it is; but I take it that the whole pith of the theory is that there is a special micro-organism, which may be for all we know very rare. Nor is it essential to the theory that there should be gastro-intestinal lesions, for although a diseased intestine forms a favorable seat for the special micro-organisms, yet it is conceivable that they may, if exceptionally numerous or vigorous, grow on healthy intestines. Dr. Halliburton[1] points out a flaw in one part of Dr. Hunter's chain of evidence, for he says that "Hunter gives no proofs that the yellow globules in the kidney consist of hæmoglobin or are derived from it; they may, indeed, be often found in perfectly normal kidneys, and give none of the reactions of hæmoglobin."

These recent theories demand that for the future attention should be directed to the following points:

(1) The frequency of vomiting, diarrhœa, and other gastro-intestinal symptoms.

[1] 'Text-book of Chemical Physiology and Pathology,' p. 301.

(2) The presence at the post-mortem examination of any gastro-intestinal lesions.

(3) Toxic symptoms.

(4) The amount of iron in the liver and other organs.

(5) The colour of the urine, and the determination of the cause of this colour.

(6) The pigment granules seen in renal cells in the urine.

(7) The investigation of such granules in the kidney after death.

(8) The colour of the fæces.

(9) Aromatic bodies in the urine.

(10) Ptomaines in the urine.

The frequency of diarrhœa, vomiting, and other gastro-intestinal symptoms.—Addison[1] said that the subjects of pernicious anæmia had not previously suffered from "exhausting diarrhœa." Bristowe[2] says, "The digestive system commonly suffers, the appetite usually fails, dyspeptic symptoms with sickness after food are of frequent occurrence, and as a general rule the bowels are constipated." Dr. Pye-Smith[3] states that "if we admit the secondary form of progressive anæmia as the 'same disease' with the primary," then according to Eichhorst, in twenty-four out of sixty-two cases dyspepsia and diarrhœa were predisposing causes. Fagge says, "In another group of cases of grave or even fatal anæmia the origin of the disease was in protracted diarrhœa or in gastric ulceration."[4] From the wording of this sentence it is left an open question whether these were cases of pernicious anæmia as described by Addison. Byrom Bramwell[5] says, "In more than one other case of profound anæmia which presented many of the symptoms of pernicious anæmia I have met with obstinate and long-continued diarrhœa. In some cases of genuine pernicious anæmia diarrhœa does occur." Dr. F. Taylor[6] says the gastro-intestinal disturb-

[1] Op. cit.

[2] 'Brit. Med. Journ.,' 1888, p. 1149.

[3] 'Guy's Hosp. Rep.,' series 3, vol. xxvi, p. 267.

[4] 'Principles and Practice of Medicine,' vol. ii, p. 774.

[5] 'Studies in Clin. Med.,' p. 30.

[6] 'The Practice of Medicine,' pp. 662 and 663.

ance has been credited with causing pernicious anæmia, but in describing the symptoms he states that "dryness of the mouth and throat, loss of appetite, nausea, and generally constipation are present." Coupland, on the other hand, says, "Frequently vomiting and diarrhœa are early symptoms."[1] In the face of such divergence of opinion, and because of the theoretical importance of gastro-intestinal symptoms, an examination of the cases which are appended to this paper should be of value. It is particularly to be remembered that in all the diagnosis was allowed to be correct even after post-mortem examination, and that those who had charge of the cases were either pupils of Addison himself or of his pupils.

The following is an abbreviated account of the gastro-intestinal symptoms in each case.

Case 1.—Before admission, none. After admission, frequent vomiting and diarrhœa.

Case 2.—Before admission, none. After admission, none.

Case 3.—Before admission, lately had had diarrhœa. After admission, often vomiting, at times diarrhœa.

Case 4.—Before admission, none. After admission, bowels at first regular or constipated, frequently very much relaxed for the last six weeks.

Case 5.—Before admission, for some weeks constant diarrhœa and vomiting. After admission, vomiting and very severe diarrhœa.

Case 6.—Before admission, none. After admission, repeated vomiting, no diarrhœa.

Case 7.—Before admission, none. After admission, none

Case 8.—Before admission, subject to diarrhœa. After admission, none.

Case 9.—Before admission, vomiting, occasional for many years, daily for last eight months. Sternal pain and burning sensation in back; alternating constipation and diarrhœa. After admission, sometimes vomiting and pain.

Case 10.—Before admission, none. After admission, none.

Case 11.—Before admission, none. After admission, bowels confined.

[1] Fowler's 'Dictionary of Practical Medicine,' p. 47.

Case 12.—Before admission, one attack of severe diarrhœa and vomiting. After admission, bowels confined.

Case 13.—Before admission, frequently vomited. After admission, vomiting and constipation.

Case 14.—Before admission, nausea and vomiting. After admission, coppery taste in mouth.

Case 15.—Before admission, failing appetite, occasional diarrhœa. After admission, none.

Case 16.—Before admission, a week's bad diarrhœa. Loss of appetite. After admission, tongue furred. Appetite bad.

Case 17.—Before admission, none. After admission, vomiting, bowels relaxed.

Case 18.—Before admission, diarrhœa and vomiting. After admission, vomiting and abdominal pain. Diarrhœa last two days.

Case 19.—Before admission, loss of appetite, epigastric pain, vomiting, flatulence. After admission, diarrhœa, pain, vomiting, and loathing of food.

Case 20.—Before admission, retching and vomiting. After admission, tongue furred, raw, fissured; no vomiting.

Case 21.—Before admission, none. After admission, none.

Case 22.—Before admission, none. After admission, none.

Case 23.—Before admission, attacks of epigastric pain, loss of appetite. After admission, much vomiting and diarrhœa.

Case 24.—Before admission, severe diarrhœa. After admission, constipation.

Case 25.—Before admission, dyspepsia. After admission, constipation, abdominal pain, occasional vomiting.

Case 26.—Before admission, always been troubled with diarrhœa, and last two years vomiting and pain also. After admission, none.

Case 27.—Before admission, much vomiting and abdominal pain. After admission, almost constant vomiting, diarrhœa, and pain.

Case 28.—Before admission, vomiting. After admission, very sick.

Case 29.—Before admission, dyspepsia and vomiting. After admission, occasional diarrhœa, vomiting, and flatulence.

Case 30.—Before admission, none. After admission, a little diarrhœa.

Case 31.—Before admission, none. After admission, diarrhœa and vomiting towards the end of the case.

In analysing these symptoms it must be remembered that unless attention is especially directed to any point it is often omitted from a clinical report, and that the previous histories given by patients are often very imperfect, so that it is likely that these symptoms occurred in a larger percentage of cases than these figures actually represent. Cases 21 and 22 had better be omitted from consideration, for there is no clinical report of the latter, and the clinical report of the former consists of only two or three lines. It will be seen that twelve out of twenty-nine, or 41 per cent., gave a history of vomiting before admission; that ten, or 34·5 per cent., gave a history of diarrhœa. After admission, sixteen, or 55 per cent., suffered from vomiting; and twelve, or 41 per cent., suffered from diarrhœa. Six patients complained of constipation, seven of abdominal pain, others of loss of appetite, nausea, flatulence, loathing of food, and furred or raw tongue. I feel sure that anyone reading these figures, or the reports of the cases, will agree that these frequent and severe dyspeptic symptoms, especially the vomiting and diarrhœa, cannot be explained by supposing them to be due to the anæmia from which these patients suffered; nor can they be attributed to the arsenic which was often administered, for they were present in cases in which this drug was not used, many patients continued to suffer from vomiting and diarrhœa after discontinuing the arsenic, and others suffered before they took any arsenic.

We may therefore conclude that dyspeptic symptoms, particularly vomiting and diarrhœa, are very common in genuine pernicious anæmia, being present in almost half the cases, that they are often very severe, and that constipation is one of the least common of the dyspeptic symptoms.

The presence at the post-mortem examination of any gastro-intestinal lesions.—This is a very important question, for the disease most frequently mistaken for pernicious anæmia is malignant disease of the stomach; and Dr. Fenwick has described a form of anæmia which is very like pernicious

anæmia, and which is associated with atrophy of the gastric glands.

Among the cases here recorded it is expressly stated that the stomach and intestines were absolutely healthy in Cases 5, 7, 11, 12, 16, 17, 23, 27, 28, 30, and 31. In Cases 18 and 20 the appearances described are often met with in healthy intestines; and in Cases 1, 2, 4, 6, 8, 9, 10, 14, 19, 21, 22 the gastro-intestinal tract may be taken as healthy to the naked eye, for the autopsy was made and recorded very carefully, and yet no mention is made of the stomach and intestines.

In the following cases, however, something abnormal was found. In Case 3 the large intestine contained cicatrices of healed ulcers and a little diphtheritic exudation. In Case 13 there were one or two small blackish spots on the mucous membrane of the cæcum, almost "diphtheritic-looking." In Case 15 the mucous membrane of the stomach, especially the pylorus, was thickened. In Case 24, a foot above the cæcum there was a cicatrix of a small healed ulcer. In Case 25 there were a few follicular ulcers of the colon, and in Case 29 the lower part of the small intestine showed several scars. In none of these cases is there any record of a microscopical examination of the stomach and intestines. Thus in six cases out of thirty-one naked-eye changes were found in the gastro-intestinal tract, but they were all slight. In all six cases either vomiting or diarrhœa or both had been present during life, but not always severely, and in many cases in which they were very severe no changes were found in the stomach or intestines. In Dr. Hunter's case the microscope revealed acute and subacute gastritis, but in Dr. Mott's third case, although microscopical sections were made, the stomach and intestines were normal. Occasionally the mesenteric glands are slightly enlarged, but not more than the irritation of the diarrhœa will explain.

It is impossible to say in the present state of our knowledge whether these slight lesions are of much importance, but it is clear that for the future attention must be directed to this point.

Toxic symptoms.—We have seen that the new theory of the pathology of pernicious anæmia attaches much importance to these. Whenever the report mentions them I have included them in the account of the cases which are appended to this paper. Case 4, a few days before death, lay in a half-conscious state, and at night he was delirious. Case 12 had four fits, during which he was unconscious for five minutes. Case 13 was, on May 8th, awaked early in the morning by shivering, which was soon followed by profuse sweating. Temp. 104·6°. Tongue dry and brown. She had other rigors before she died on May 23rd. Case 14 complained of drowsiness. Case 16 was a little drowsy on one occasion. Case 20 was on October 21st drowsy and tired, and hardly in his right senses. On October 23rd he was delirious and violent, and had to be confined to the strong-room, and on October 24th he was semi-comatose with contracted pupils, and was occasionally delirious. Case 21 was semi-comatose shortly before death. Case 22 became comatose before death. Case 23 was drowsy before death. Case 24 often had bad headaches, during which he perspired freely and breathed irregularly. Case 30 had an epileptiform attack, after which he was dull for some days.

It is well known that coma and even delirium may result from extreme anæmia, and therefore it is doubtful whether much stress should be laid upon these symptoms in Cases 4, 14, 16, 21, 22, and 23. In Cases 12 and 30 the fits may have been accidental. Case 20 had drunk a good deal. Case 24 had headaches, but they are common in anæmia; however, it is just possible the sweating may have been toxic, and in Case 13 the symptoms may have been toxic. The result, then, of our examination of these cases is that on the whole the evidence of toxæmia is slight.

The presence of iron in the liver and spleen.—In Case 29 an analysis was made by Dr. Stevenson, and he found that the dried liver contained 0·37 per cent. of iron, and the dried spleen 0·184 per cent. We have seen that ordinarily the dried liver contains 0·083 per cent. of iron, and the dried spleen 0·171 per cent., and that in perni-

cious anæmia the quantity of iron in the liver may be increased sevenfold, and that the proportion of iron in the liver to that in the spleen, which is ordinarily as 1 is to 2, is increased. These facts are borne out by Dr. Stevenson's analysis, which shows the iron in the liver to be four and a half times as much as normal; and the proportion of iron in the liver to that in the spleen is two to one—that is, it is increased fourfold. Thus the chemical analysis in this case quite supports the previous analyses.

The colour of the urine.—The urine is described as high-coloured or dark at some period or another of the illness in Cases 4, 14, 18, 20, 23, 26, and 29. In Case 20 on the first admission it was dark, but contained a trace of bile; on the second admission it was dark, apparently without any bile. In Case 13 it is noted that it gave a dark red colour with nitric acid; and in Case 14 it was observed that the dark colour became darker still on the addition of nitric acid, and the same result was obtained in Case 23. In Case 19 it is described as yellow. In Case 30 it is said to be light in colour, and in Case 31 to be normal. The urine is therefore stated to be dark in seven out of thirty-one cases, or, as two at least are very imperfect in their clinical details, in seven out of twenty-nine, that is nearly 25 per cent. But this clearly very much understates the proportion, for the colour of the urine is often omitted from the report, and in no case is there a daily examination of the colour. We may therefore, I think, conclude that probably in pernicious anæmia the urine is frequently but not always high-coloured, and this conclusion agrees with the opinion expressed by Dr. Stephen Mackenzie.[1] In none of the cases is it stated to what the high colour is due, but in one patient who is still alive, and therefore whose case is not published here, the urine, which Dr. MacMunn was kind enough to examine for me, contained normal and not pathological urobilin; but it should be stated that it was not very dark in colour, and the patient was recovering.

The records at Guy's Hospital unfortunately do not give any statements about the pigment granules in the urine or

[1] 'Brit. Med. Journal,' January 24th, 1891, p. 167.

kidneys, nor about ptomaines or aromatic bodies in the urine, nor about the colour of the fæces.

It is worth while to note that in several cases the bones were examined, but nothing abnormal was discovered.

Summary.—Among the thirty-one cases in Guy's Hospital which have died during thirty-four years, dyspeptic symptoms, especially diarrhœa and vomiting, which may be very severe, are quite common; high-coloured urine is sometimes, and probably frequently, present; there is not much evidence of toxic symptoms; slight intestinal lesions are occasionally found, but not sufficiently often for it to be certain that they have much value; and the only case analysed showed that the quantity of iron in the liver, and the proportion of iron in the liver to that in the spleen, were both in excess of the normal.

Part II.—The Prognosis of Pernicious Anæmia.

Addison[1] referred to the possibility that these patients may recover; for he said, "With perhaps a single exception, the disease, in my own experience, resisted all remedial efforts, and sooner or later terminated fatally."

Dr. Pye-Smith[2] collected twenty cases of recovery from "idiopathic and profound anæmia," and recorded an important case of a man aged fifty-three who suffered from pernicious anæmia, and was treated by arsenic in 1880; he improved, and in 1882, when the report of his case was published, he was still quite well.[3]

[1] Op. cit.

[2] Op. cit.

[3] Dr. Pye-Smith has kindly supplied me with the following notes of this patient's condition since 1882:

July 8th, 1885.—The patient is stout and in good health, he has no anæmia; the blood examined under the microscope is normal. The seat of the retinal hæmorrhage is still evident.

October 13th, 1886.—Occasionally his eczema reappears. He is well nourished, but pale. His gums and lips are of good colour. The pulse is normal; there is no pyrexia. There is some dyspnœa on going upstairs.

The patient passed under the care of Dr. Uhthoff, of Brighton, who, on March 7th, 1889, wrote to Dr. Pye-Smith to say that he had some œdema

Dr. Broadbent[1] records the case of a woman aged twenty-six who showed all the signs of pernicious anæmia; she was in the hospital from February 7th to May 20th; during this time she took arsenic. She was heard of two months after leaving the hospital, and she was then well.

Dr. Bristowe says, "Undoubtedly there are many cases in which a more or less perfect recovery takes place—a recovery, however, attended with a liability to a relapse."

During the ten years 1879—1889 there have been in Guy's Hospital ten patients who have been thought, while in the hospital, to be suffering from pernicious anæmia, but who have been discharged. One of these was under my care, but at his own request he was sent home shortly after admission, and I have since heard that he died three months after leaving us. Another one, Richard P—, also under my care, will be presently described in detail, as he is still alive. The remaining eight were all discharged as improved, and have not returned to the hospital; one of these eight gave no address. I have sent a reply post-card to each of the remaining seven. Six of the seven post-cards have been returned marked by the post office authorities to say that the address is unknown. The friends of the seventh, W. K—, who was admitted under Dr. Fagge in 1881, write to say that he died on October 26th, 1884, and that he did not get well for long together, but was usually weak and feeble, and attended Brompton Hospital as an out-patient.

We thus see that, with one exception, there is no evidence that any of the cases which have been discharged from Guy's Hospital during the last ten years as having improved have completely recovered for at least any length of time; and as six could not be found, and the seventh has died, the strong probability is that most have died.

of the legs and a trace of albumen, but on March 24th, 1889, he was recovering.

I wrote to Dr. Uhthoff about this patient, and he kindly told me that he had been under his care from time to time with anæmia, but that he had benefited by arsenic.

At the present time, January, 1891, he is being attended by Mr. H. H. Taylor (Dr. Uhthoff's partner), of Brighton, who kindly writes to say that now he is recovering from a slight attack of bronchitis. He is of good colour and fairly stout.

[1] 'Lancet,' vol. i, 1889, p. 75.

Among the cases in the appendix to this paper, Case 3 improved sufficiently to leave the hospital in April, but he returned the next August, and soon died. Case 9 got very much better; he was able to leave on February 1st, but he returned on October 4th of the same year, and died December 14th. Case 23 was very ill and had severe diarrhœa, but he improved very much, and was discharged on July 29th; but at the end of the same year he was readmitted suffering from the same malady, and he died on May 13th of the next year. Case 29 left the hospital on January 27th; she remained well for some time, but was readmitted June 12th. Again she improved, and left on August 7th, but was re-admitted October 29th, and died November 20th. Case 31 was in the hospital in 1887 for pernicious anæmia, and he improved very much and went out. On March 5th, 1889, he was readmitted for pernicious anæmia; he improved and went out on March 13th, but on June 5th he was again admitted for the same disease, and he died on June 30th. This case shows that even as long as two years after apparent recovery the disease may break out afresh and kill.

I have not alluded to Case 20, because it is by no means certain that the illness for which he was previously in the hospital was pernicious anæmia; nor to Case 19, because we only have the patient's word for it that his previous illness was like that from which he died. Cases 23, 29, and 31 were all treated by arsenic.

Now it is a mere chance that Cases 3, 9, 23, 29, and 31 happened to return to the same hospital; if they had not they would have remained in our records as cases of pernicious anæmia cured, although they all died from the disease. This, taken with the fact that, with one exception, I have not been able to get any evidence that any of the cases which during the last ten years have been discharged as improved have remained well, shows that the prognosis is very gloomy.

A case recorded by Dr. Tyrrell-Brooks[1] also shows the importance of following up patients supposed to be cured from pernicious anæmia. A woman aged thirty-eight was admitted under his care for pernicious anæmia, December 27th, 1884.

[1] 'Lancet,' 1887, vol. ii, p. 859.

She was treated with arsenic, and was discharged as cured on February 25th, 1885; but the disease returned, and she died in January, 1886.

Taking together all the fatal cases here mentioned, W. K— died three years after he was first admitted with pernicious anæmia, Case 3 died nine months after his first admission, Case 9 died fifteen months afterwards, Cases 23 and 29 each died eleven months afterwards, and Case 31 died two years afterwards. Dr. Tyrrell-Brooks's case died one year afterwards. W. K— was ill during most of the three years which intervened between the first attack and his death. It is stated of Case 3 that soon the diarrhœa and epistaxis returned. Out of the fifteen months that Case 9 lived she was ill enough to be in the hospital for seven months. Case 23 left the hospital in July, but the symptoms returned in October. Case 29 returned to the hospital on the first occasion in four months and a half, and on the second occasion the symptoms returned six weeks after the patient's discharge. In Case 31 the patient remained apparently well for about two years. In Dr. Tyrrell-Brooks's case the interval between her discharge from the hospital and her death was eleven months, so the interval between her discharge and the relapse must have been less.

We thus learn that although several patients have been known to recover from their first or even their second attack, nevertheless even then almost invariably they soon relapse and die, and that generally within a year. Most of the cases that have been benefited have taken arsenic.

This result clearly shows that for the future cases should not be recorded as having recovered unless they have been followed up for some few years at least after their apparent recovery. Dr. Pye-Smith's case already alluded to fulfils these conditions, but Dr. Broadbent's case does not, for we are only told the condition of the patient two months after leaving the hospital. The subsequent history of the "Twenty Cases of Recovery from Idiopathic and Profound Anæmia," collected by Dr. Pye-Smith, would be very valuable. I strongly suspect that many of the patients, if they were really suffering from pernicious anæmia, would have been found to have died soon after treatment had been dis-

continued; in fact, it is stated that one of Biermer's did die a year after her discharge.

The following remarkable case seems to me worthy of record, for it is an example of a patient who was in Guy's Hospital in July, 1887, for pernicious anæmia; he completely recovered, and now, nearly four years after his coming into the hospital, he is as well as can be. I often see him; he shows no signs of anæmia, and appears in excellent health. It does not seem to me likely that there can have been a mistake in diagnosis, for not only did he, in the condition of his blood, his colour, his failure to improve upon iron, and his rapid improvement upon arsenic, show the chief symptoms of pernicious anæmia, but the two maladies with which this disease is commonly confounded are phthisis—from which the patient could hardly have recovered so rapidly had it been severe enough to make him as ill as he was—and malignant disease of the stomach or intestine, which must have been fatal long before now.

In Dr. Pye-Smith's patient, whose case is given on p. 168, and this patient, the recovery is so complete and has lasted so long that they may fairly be said to be cured. They both took arsenic, and were both men over forty. I believe there are no other instances on record of recovery lasting so long.

Case of recovery from pernicious anæmia.—Richard P—, æt. 42, of 49, Abbeyfield Road, Rotherhithe, a butcher, was admitted into Guy's Hospital July 6th, 1887. The family history and previous history are unimportant. In March he felt his legs and back gradually getting weak, so that he was unable to work as long as usual; this he is inclined to attribute to a fall on some ice, but fourteen days intervened between the fall and the beginning of his illness. A month after, he was so weak and had such numbness in the legs that he lay in bed for a week; when he got up he could only walk with a stick. During the summer he had frequently had to lie up for a day or two on account of this weakness. In June he had an attack of what he calls erysipelas, but this got quite well. He has suffered from dyspnœa, but never from diarrhœa or sickness, nor has he ever noticed his water to be high-coloured.

Condition on admission.—Fairly well nourished, but is everywhere of a primrose colour. The left leg is somewhat swollen, and has a medium-sized eczematous ulcer on it. Alimentary system normal. Circulatory system: There is a soft systolic bruit both at the apex and the base. P. 96. Respiratory system normal. Urine normal. Nervous system: Walks in a very staggering manner, but he is very weak muscularly. The reflexes and electrical reactions are all normal, and there is no evidence of any disease of the nerves. Optic discs normal.

Blood.—No leucocytosis. Number of red corpuscles reduced to 23 per cent. of the normal number; some of them are rather irregular in shape.

The most thorough scrutiny failed to detect any cause for the anæmia.

First of all the patient had five-grain doses of reduced iron three times a day; after a fortnight of this treatment it was found that the number of red corpuscles was exactly the same.

He was next put upon arsenic, taking five minims of Fowler's solution three times a day; at the end of a week the number of corpuscles remained the same, and the patient's colour was the same as on admission, although he said he felt stronger. After taking the arsenic another week an erythematous eruption, consisting of patches about as large as a threepenny piece, appeared on the face and neck; this lasted for about a fortnight. Its departure did not seem to be accelerated by the stoppage of the arsenic, which was accordingly resumed. The colour and strength of the patient gradually improved, so that when he left the hospital on September 22nd he was of a fairly healthy colour, and could walk with hardly any difficulty, and was in every way stronger. The number of corpuscles gradually increased till, when he went out, they were 80 per cent. of the normal number.

October 22nd, 1889.—I examined the blood of this patient; corpuscles (red) 54 to the square (108 per cent.), white normal, hæmoglobin 115 per cent. of normal. Is stout, in excellent health; no murmur, no optic neuritis, discs normal.

February 21st, 1891.—Saw him to-day; he is in robust health, good colour. Never has diarrhœa nor sickness. Has not noticed that his urine is high-coloured.

Appendix of Cases of Pernicious Anæmia which have died in Guy's Hospital from 1855 to 1889, both Years inclusive.

Case 1. (Recorded by Dr. Wilks, 'Guy's Hospital Reports,' series 3, vol. iii, p. 205.)—Mary B—, æt. 31, admitted under Dr. Hughes March, 1855. For a year her health had been failing, but she denied having had any hæmorrhage, diarrhœa, or any other symptoms excepting those dependent upon her gradual increasing debility.

On admission she grew feebler and feebler until her death, a fortnight after admission. During this time she had frequent vomiting and diarrhœa.

Autopsy.—Heart presented an extreme degree of fatty degeneration; the liver was very fatty.

Case 2. (Recorded by Dr. Wilks, 'Guy's Hospital Reports,' series 3, vol. iii, p. 206.)—Martha D—, æt. 28, was admitted under Dr. Oldham November 13th, 1856. There was no history of diarrhœa.

After admission she daily became weaker without any marked symptoms of derangement of any particular organ.

Autopsy.—The heart had undergone extreme fatty degeneration. The liver was also very fatty.

Case 3. (Recorded by Dr. Wilks, 'Guy's Hospital Reports,' series 3, vol. iii, p. 207.)—William S—, æt. 16, was admitted under Dr. Addison in January, 1856. He had not been well for three years, and had often suffered from bleeding at the nose, and lately had had diarrhœa.

After admission into the hospital he often had sickness and sometimes diarrhœa. In April he began rapidly to improve, and he left the hospital. He went to Berkhampstead, and was for a time better, but soon the diarrhœa and epistaxis returned, and he came back to the hospital in August. He kept his bed, and daily sank, at times having diarrhœa and sickness.

Autopsy.—Heart in a most extreme degree of fatty degeneration. The large intestines contained numerous cicatrices of ulcers or ulcers nearly healed, and a little recent diphtheritic inflammatory exudation in parts. The lymphatic glands were healthy.

Case 4. (Recorded by Dr. Wilks, 'Guy's Hospital Reports,' series 3, vol. iii, p. 208. I have read the report in the Hospital Records.)—Charles P—, æt. 52, admitted under Dr. Barlow April 16th, 1856.

On admission tongue normal, urine straw-coloured.

April 23rd.—Bowels regular, urine normal in colour, tongue normal.

28th.—Vomits in the morning, tongue normal, appetite good.

May 19th.—Bowels confined, urine normal.

June 9th.—Urine rather high-coloured. He has had a pain in the umbilical region, but he has had purgatives, and these will probably explain it.

19th.—Urine clear.

30th.—Urine normal in colour, tongue normal.

July 7th.—Bowels very much relaxed.

11th.—Bowels very much relaxed.

12th.—Bowels less relaxed, urine not so high-coloured.

14th.—Bowels still relaxed.

August 4th.—Bowels very much relaxed.

11th.—Bowels very much relaxed. He died a day or two after this date. A few days before death he lay in a half-conscious state, and at night he was delirious.

Autopsy.—Heart and liver both very fatty.

Case 5. (Recorded by Dr. Wilks, 'Guy's Hospital Reports,' series 3, vol. iii, p. 209. I have read the report in the Hospital Records.)—Mary Ann H—, æt. 31, admitted under Dr. Addison's care May 30th, 1853. She was in an extreme state of anæmia, and she stated that she was very ill during the last few weeks of pregnancy, having had constant diarrhœa with passage of blood, and also vomiting with occasional blood. She had now been confined three weeks.

On admission the tongue was normal; there was no diarrhœa up to June 10th.

11th.—Diarrhœa.

15th.—Diarrhœa.

18th.—Diarrhœa, sickness, and abdominal pain.

21st.—Much vomiting. Diarrhœa, the bowels open six times in twenty-four hours.

22nd.—Bowels still relaxed.

23rd.—Bowels open eight times in twenty-four hours; vomiting.

25th and 26th.—Diarrhœa continues.

27th.—Bowels open twenty or thirty times in the night.

28th.—Bowels still relaxed.

29th.—Death. The urine is mentioned, but its colour is not stated.

Autopsy.—Heart and liver very fatty. Stomach and intestines presented a healthy appearance, no trace of ulceration in any part of their course.

Case 6. (Recorded by Dr. Wilks, 'Guy's Hospital Reports,' series 3, vol. iii, p. 210.)—Eliza T—, æt. 50; she had been an inmate of the lunatic ward for thirteen years. During the last few months of her life she became gradually very weak and anæmic, and had repeated vomitings. She had no diarrhœa.

Autopsy.—Heart, liver, and kidneys very fatty.

Case 7. (Recorded by Dr. Wilks, 'Guy's Hospital Reports,' series 3, vol. iii, p. 211.)—Admitted under Dr. Hughes April, 1854. He had been gradually getting weak for four months, and two weeks before he passed a large quantity of blood at stool.

On admission no mention is made of any diarrhœa or sickness. He gradually sank, and died three weeks after admission.

Autopsy.—The heart, liver, and kidneys were very fatty. There was no ulceration in the intestines.

Case 8. (Recorded by Dr. Wilks, 'Guy's Hospital Re-

ports,' series 3, vol. v, p. 108.)—A. W—, æt. 46, was admitted under Dr. Barlow December 21st, 1858. For the last three years she has been getting weak, and has been subject to diarrhœa.

On admission she gradually became more and more feeble. There is no mention of diarrhœa or sickness during her stay in the hospital. She died seven weeks after admission.

Autopsy.—Heart and liver fatty, lungs œdematous.

Case 9. (Recorded by Dr. Habershon in the 'Lancet,' 1863, vol. i, pp. 518, 551; and by Dr. F. Taylor, 'Guy's Hospital Reports,' series 3, vol. xxiii, p. 189.)—Eliza W—, æt. 40, admitted September 25th, 1861. She had for many years suffered from occasional attacks of vomiting, but for the last eight months these had been of almost daily occurrence, with an occasional intermission of a week. The vomiting often came on directly after food; at other times there was an interval of some hours. There was pain down the sternum of an aching character. This was increased by food; a burning sensation in the back and shoulders troubled her. The bowels were sometimes much relaxed, at other times confined for several days together. Tongue clean.

September 28th.—Complains of much sickness and pain.

October 19th.—Had an attack of severe abdominal pain and prostration.

January 1st, 1862.—Better.

February 1st.—Much improved, enjoys food. Left the hospital.

Readmitted October 4th, 1862. Blanched, emaciated, and feeble. Gradually sank, and died December 14th.

Autopsy.—Fatty heart.

Case 10. (Recorded by Dr. F. Taylor, 'Guy's Hospital Reports,' series 3, vol. xxiii, p. 190.)—Henry R—, æt. 22, admitted under Dr. Barlow November 30th, 1864. A month ago he began to get weak.

On admission tongue white and furred. No mention of diarrhœa.

He died December 8th.

Autopsy.—Except for fatty change the organs appeared quite healthy.

Case 11. (Recorded by Dr. F. Taylor, 'Guy's Hospital Reports,' series 3, vol. xxiii, p. 192. I have read the report in the 'Hospital Records.')—George B—, æt. 51, was admitted under Dr. Wilks June 27th, 1868.

July 11th.—It is noted that the bowels have been very confined, and the patient has often been sick. The urine is mentioned, but its colour is not stated.

Autopsy.—The stomach was normal, and it is especially stated that there was no trace of disease in all the length of the bowel.

Case 12. (Recorded by Dr. F. Taylor, 'Guy's Hospital Reports,' series 3, vol. xxiii, p. 194. I have read the report in the 'Hospital Records.')—George H—, æt. 68, admitted under Dr. Wilks May, 1870. Quite well till eighteen months ago, when he had diarrhœa and vomiting, lasting two or three days, and leaving him very weak. But he regained strength and remained well for a month or so, when his appetite began to fail. Five months ago he had a fit, during which he was unconscious for about five minutes; he has had three similar attacks since.

On admission the urine was high-coloured, sp. gr. 1015; no albumen. On May 7th it is stated that the bowels were very confined. He gradually sank, and died on June 18th.

Autopsy.—The heart showed well-marked fatty degeneration. The stomach and intestines were quite healthy.

Case 13. (Recorded by Dr. F. Taylor, 'Guy's Hospital Reports,' series 3, vol. xxiii, p. 199. I have read the report of this case in the 'Hospital Records.')—Margaret M—, æt. 32. Six months ago ceased to menstruate, and began frequently to be sick; no mention in the report of pregnancy.

On admission, on April 29th, 1874, she was vomiting. The urine gave a deep red colour with nitric acid, otherwise it was normal.

May 1st.—Patient was sick.

5th.—Urine not so pale as on admission.

8th.—Early this morning awakened by shivering, which was soon followed by profuse sweating. Her temperature rose to 104·6°. The tongue was dry and brown, and she had noises in the head. 3 p.m., temp. 101·1°; 5 p.m., temp. 102·8°; 10 p.m., temp. 101°.

9th.—Better this morning. Temp. 101°. Urine pale, gives a very slight colour with nitric acid. Bowels very constipated. She died May 26th. She had other rigors before she died.

Autopsy.—Heart very fatty. Stomach, ileum, mesenteries, and lumbar glands all healthy. Cæcum œdematous; one or two blackish small spots on the mucous membrane, almost diphtheritic-looking.

Case 14. (Recorded by Dr. F. Taylor, 'Guy's Hospital Reports,' series 3, vol. xxiii, p. 201. I have read the report of this case in the 'Hospital Records.')—William C—, æt. 47, was admitted under Dr. Wilks August 18th, 1874. Ten months ago nausea and sickness set in, and the patient had to go to bed. After four months' treatment he improved sufficiently to get about his work again, but soon he again became too weak.

On admission he complains of a coppery taste in the mouth and a feeling of drowsiness. The urine is dark in colour, and becomes darker on the addition of nitric acid. He improved at first, but gradually sank, and died September 16th.

Autopsy.—Nothing noteworthy, save that all the organs were anæmic.

Case 15. (Recorded by Dr. Taylor, 'Guy's Hospital Reports,' series 3, vol. xxiii, p. 202; and Dr. Pye-Smith, 'Virchow's Archiv,' 1875, Bd. lxv, p. 510.)—J. K—, æt. 52. He was admitted under Dr. Pye-Smith in December, 1874. He had complained some months of failing appetite, weakness, and occasional diarrhœa. He died January 23rd, 1875.

Autopsy.—Heart, fatty degeneration. The mucous membrane of the stomach, especially of the pylorus, was thickened.

Case 16. (Recorded by Dr. Taylor, 'Guy's Hospital Reports,' series 3, and Dr. Pye-Smith, 'Virchow's Archiv,'

1875, Bd. lxv, p. 511. I have read the report in the 'Hospital Records.')—William S—, æt. 47, was admitted December 23rd, 1874. Five or six months ago he was in bed for a week with bad diarrhœa. He got to work again after this, but he has felt weak and suffered from loss of appetite ever since.

January 3rd.—It is noted that his tongue is furred and his appetite bad.

14th.—He was a little drowsy. He obtained temporary relief, but relapsed, and died June 21st, 1875.

Autopsy.—Heart and liver showed fatty degeneration. Stomach and intestines healthy.

Case 17. (Recorded by Dr. Taylor, 'Guy's Hospital Reports,' series 3, vol. xxiii, p. 202; and by Dr. Habershon, 'Medical Times and Gazette,' 1876, vol. i, p. 249. I have read the report in the 'Hospital Records.')—Leigh E—, æt. 21, was admitted under Dr. Habershon January 28th, 1876. For some time past he has been getting weak, but his bowels and appetite have been regular. There has not been any vomiting.

On admission the urine contained a quantity of lithates.

February 8th.—He was sick, and the bowels were relaxed.

9th and 10th.—He was sick. He gradually sank, and died February 11th. Transfusion was tried.

Autopsy.—Heart, fatty degeneration. Stomach and intestines healthy.

Case 18. (Recorded by Dr. Taylor, 'Guy's Hospital Reports,' series 3, vol. xxiii, p. 207. I have read the report in the 'Hospital Records.')—May H—, æt. 15, was admitted under Dr. Habershon November, 1876. She had had diarrhœa from time to time before admission. She has also had vomiting.

On admission the urine was high-coloured.

November 16th.—Sickness this morning.

December 1st.—There has been much abdominal pain and sickness during the last few days.

3rd.—Bowels open three times a day.

4th.—Bowels relaxed; continual vomiting. On this day she died.

Autopsy.—Heart very fatty. In the intestine Peyer's patches were very plainly marked. The mesenteric glands were large.

Case 19. (Recorded by Dr. Taylor, 'Guy's Hospital Reports,' series 3, vol. xxvii, p. 204; and by Messrs. Mackern and Davy, 'Lancet,' May, 1877. I have read the report in the 'Hospital Records.')—John B—, æt. 53, was admitted under Dr. Moxon September 20th, 1876. Ten years ago he had an attack much like the present; he became weak; he had fainting fits; he was yellow in colour, very ill and delirious for fourteen days. He lay up for two months. He has not been strong since, suffering from breathlessness, palpitation, loss of appetite, pain in the epigastrium, sickness, and flatulence.

On admission the bowels were very irregular—he had constipation alternating with diarrhœa. The urine was yellow.

October 11th.—Diarrhœa three or four times a day.

15th.—Diarrhœa three or four times a day.

27th.—Abdominal pain and nausea.

28th.—Sickness and diarrhœa still severe; appetite very bad.

30th.—Loathes his food.

November 2nd.—Vomiting.

8th.—Vomiting and diarrhœa have stopped. He sank, and died November 21st.

Autopsy.—Heart, fatty degeneration.

Case 20. (For the notes of this case I am indebted to Dr. F. Taylor. It has not previously been published, but is Case 101 in Dr. Pye-Smith's table in the 'Guy's Hospital Reports,' series 3, vol. xxvi, p. 303. I have read the report in the 'Hospital Records.')—George M—, æt. 38, was admitted under Dr. Habershon October 19th, 1877.

He is a married man, and has four healthy children. At the age of twenty-one he went to China in the Peninsular and Oriental Company's service, and there had a slight sunstroke. Shortly afterwards he went with his ship to India, but was invalided home for dysentery after three years' service. The dysentery continued for eighteen

months or two years. He finally went to Turkey, and the dysentery ceased six or eight months after his arrival there. He was in Turkey ten and a half years, and whilst there he continued the practice, begun in India, of drinking six or eight glasses of spirit and water daily. All this time he was subject to morning retching. On getting up he would feel sick, and bring up frothy fluid; he would have little appetite for breakfast, but would improve in this respect during the day. He left Turkey a year ago; retching and vomiting have continued, and one month ago he was obliged to give up work. Since then the retching has become most violent and frequent, and the attempt to take food is followed by actual vomiting. It is somewhat doubtful whether he ever passes any altered blood, either by vomiting or by stool. During the last twelve months he has lost much flesh.

On admission he is a tall, well-built man, with a thin face. He is very anæmic and much jaundiced, the complexion and conjunctivæ being of a marked yellow tinge. The tongue is moist, pale, flabby, broad, and covered with a slight fur. On examining the chest, precordial dulness is found extending from the third to the fifth intercostal space; the heart sounds are normal; there is a blowing bruit in both jugular veins. Lungs: Right apex duller than the left; a small amount of crepitation at the end of inspiration; expiratory murmur faint. Left apex, good resonance; marked expiratory murmur; occasional sibilant râles.

The hepatic dulness appears to commence at the fourth rib in the nipple line, and to extend two inches below the ribs. There is much tenderness below the ensiform cartilage. Splenic dulness slightly increased, but the organ cannot be felt. There is no ascites and no anasarca. Urine high-coloured, with distinct evidence of bile-pigment. Sp. gr. 1015; no albumen, no sugar. Temp. 100·4°, pulse 100. Ordered—

℞ Bismuth. Carb. gr. v,
Sodæ Bicarb. gr. x,
Sp. Chloroformi ♏x,
Aquæ ℥j. Ter die sum., c̄ Succi Limonis ℥ij.

October 21st.—Passed a restless night, and was disturbed

twice to pass a motion. The nurse says that during the night he was not quite in his right senses, not understanding what was said to him, yet hardly delirious or maniacal. He is now drowsy and tired, taking but little notice of anyone, but answering questions quite well when roused. Urine contains bile-pigment; sp. gr. 1015; no albumen nor sugar.

22nd.—Passed a quieter night, but is absent-minded this morning. He is thirsty, the tongue presenting patches of brown fur. The crepitation at the right apex is less marked. There is tenderness on percussion over the enlarged liver, which appears to reach the umbilicus. The pulse is weak, and there is a loud systolic murmur over the aortic valves. The motions are free from mucus or pus. Two grains of quinine were added to each dose of the mixture.

23rd.—Passed a restless night. Tongue pale and flabby, partly furred, partly raw and fissured. He retches much less, but complains of excessive thirst, and is so deaf that it is difficult to make him understand. The pulse is remarkably small, weak, and compressible; the murmur is still present at the base of the heart; crepitation at the right apex continues. Hepatic dulness begins at the sixth rib in the middle line, and reaches to within one finger's breadth of the umbilicus. No leucine or tyrosine can be found in the urine, which is scarcely different from what it was on the 21st. In the evening about 5 p.m. he was delirious and violent, got out of bed, was anxious to fight, and put his fists through the window-panes. He was confined in the strong-room, and then became quieter; his pulse was 160 and irregular.

24th.—He passed a quiet night. This morning he is semi-comatose, with eyes open, pupils contracted, but puts out his tongue, and turns over when told; when questioned he mutters incoherent answers, but mostly takes no notice. Tongue furred, sordes on gums. Pulse 96, regular in rhythm, but unequal in force. Liver apparently smaller, reaching only within three fingers' breadth of the umbilicus. At left apex of lung bronchial breathing; at right apex the crepitation persists. Occasionally talkative and delirious.

25th.—He was delirious and restless until 3 a.m., talking incoherently and getting out of bed. His pulse last night was very irregular in strength, but mostly very feeble and quick. After 3 a.m. he slept well. This morning he seems better, his mind is clear, and he answers quite rationally. His pulse is stronger, but still irregular in volume. The tongue is cleaner; the bowels not open since the 21st. The sputum is tinged with blood; there is marked crepitation at the right apex, and bronchial breathing at the left. Systolic bruit less marked. Hepatic dulness commences at the sixth space in the mammary line; in the middle line it extends to two fingers' breadth above the umbilicus. Ordered—

℞ Pil. Col. c̄ Cal. gr. x, statim,
Haust. Sennæ ʒiss, post quatuor horas,
Mist. Effervescentis ʒj, t. d. s.

26th.—Bowels relieved. He passed a quiet night, slept well, and is much better. Tongue clean; pulse slower, firmer, and stronger. Temperature normal. Mind clear.

27th.—Removed into general ward last night. He had a prolonged sleep, and woke up much better. Tongue clean and moist; pulse of moderate strength and rapidity. Mind quite clear. Bruit less distinct; crepitation at right apex unaltered. Hepatic dulness not so marked in the middle line.

28th.—Bruit less distinct; pulse the same. Hepatic dulness extends from sixth rib to just below the costal margin. In the middle line more resonance and no tenderness.

30th.—Improvement continues. Face shows a slight flush. Bruit hardly audible; expiratory murmur now heard on left side; scarcely any crepitation at left base. Hepatic dulness begins at the sixth rib, and the organ is felt one finger's breadth below the ribs. Below the ensiform cartilage the abdomen is resonant. Tongue clean; bowels open. Was up for one and a half hours yesterday.

November 2nd.—Patient goes out in the grounds.

3rd.—Hepatic dulness from sixth rib to margin of ribs, and for two fingers' breadth below the ensiform cartilage. Slight dulness and distant crepitation at right apex; left

apex apparently normal. Heart sounds nearly normal; patient still has a sallow or earthy colour, his cheeks being at parts injected. He says he feels quite well in himself, and has a good appetite.

5th.—Discharged. The question of the case being one of pernicious anæmia had not been raised.

After his discharge he was much improved for the next month; he got rid of the jaundiced complexion, but began to suffer from ulcers in the mouth and throat, and after a time could only take fluid food. His chest became affected, and the mouth was from time to time better, but caused a good deal of irritation, which resulted in violent coughing, with scanty frothy expectoration. He was also feverish at night, and gradually grew very weak, suffering from palpitation and occasional sickness. About the third week of January, 1878, his skin was observed to be of yellow colour, and he was admitted to the hospital under Dr. Taylor's care on February 6th, 1878.

The same evening he spat up a little blood. He was then sallow, very anæmic, with a yellow tinge, which even on the conjunctivæ was very like that of jaundice. He was tall, with dark hair; fairly well developed, and not emaciated. The lungs were examined carefully with reference to the changes previously reported, but the most that could be made out was some harshness of the inspiratory and expiratory murmurs at the right apex, with mucous râles accompanying inspiration. There was little or no expectoration. The hepatic dulness was within the limits of the normal, but he had some tenderness on the epigastrium. No ascites or œdema of the lower extremities. Venous hum in the neck. Tongue dry, smooth, and flat. Urine dark in colour; albumen, perhaps a trace.

On February 13th he had been sick, and was looking more yellow. When the blood was examined, it was very pale in colour, and thin; under the microscope there was no excess of white blood-corpuscles, but a small number of the red corpuscles were altered in form, being elongated or pear-shaped, with an accumulation of colouring matter on side, resembling a nucleus, but probably due to the separation of the hæmoglobin.

February 15th.—There is increasing weakness, without other symptom than the sickness to account for it. The urine of to-day certainly contains no albumen. The optic discs are rather pale, as if from anæmia; in the fundus of the left eye there is one small hæmorrhage.

16th.—Death.

Autopsy.—Fairly nourished, but exceedingly anæmic. The brain was anæmic to an extreme degree, but otherwise healthy.

The right pleura presented some old adhesions. At the extreme apex of the right lung was some thickening, and a little nodule of calcareous matter; but no induration, nor tubercle, nor anything that could have been expected to give physical signs. The right base was highly œdematous. Both lungs, though collapsed, were markedly emphysematous at their edges. The glands at the root of the right lung were calcified. The larynx was healthy.

The heart weighed 15 oz.; its muscular fibre was firm and apparently good, but there was a just perceptible fatty striation of the musculi papillares and septum. The aortic and mitral valves were healthy.

The stomach was healthy; there were some patches of injection in the ileum, and the mucous membrane of the large intestine was irregularly injected; but there was no indication of any former dysentery.

The liver weighed 62 oz., and had the uniformly pale yellow-olive colour usual in anæmia. Its surface was smooth, its tissue free from granulation, and not at all indurated. The left lobe was quite healthy; on the surface of the right lobe were three or four whitish spots. The largest of these was apparently an encysted pentastoma. The others were all very small, not larger than millet-seeds, and some similar ones were exposed on cutting into the substance of the organ. They had orifices of ducts containing yellow bile in their centres, and appeared to be local thickenings of the walls of small bile-ducts. But they were very peculiar, and Dr. Fagge stated he had never seen anything similar.

The spleen weighed 16 oz., and was soft; the suprarenal capsules healthy. The kidneys weighed 14 oz., were anæmic, and probably slightly fatty.

Case 21. (Not published before. I have read the report in the 'Hospital Records.')—Charles S—, æt. 37, was admitted under Dr. Wilks October 2nd, 1878. He gave a history of having had epileptic fits twelve months before admission. Three weeks ago he was taken with pain all over him, and seven days ago his feet began to swell.

On admission was weak and pale; no mention of diarrhœa or sickness, but the report is very brief.

October 14th.—Being semi-comatose he was transfused, but no relief followed.

20th.—Died.

Autopsy.—Fatty degeneration of heart.

Case 22. (Not published before. I have read the report in the 'Hospital Records.')—Sarah W—, æt. 32, was admitted under Dr. Pye-Smith April 23rd, 1879.

The clinical report of this case is lost, but it appears that the patient became comatose before her death, May 5th, 1879.

Autopsy.—Fatty degeneration of the heart and œdema of the lungs.

Case 23. (Recorded by Dr. Pye-Smith, 'Guy's Hospital Reports,' series 3, vol. xxvi. I have read the report in the 'Hospital Records.')—Thomas B—, æt. 46, was admitted under Dr. Pye-Smith June 11th, 1879. Sixteen years ago he began to suffer from attacks of pain in the epigastrium and loss of appetite, sometimes with vomiting and always with a sense of languor and depression, increased by food. These attacks have recurred ever since. Sometimes their duration has been two or three days, sometimes as many weeks. He is generally troubled in this way every month. Nearly every summer he has suffered more or less from diarrhœa. During the past year the dyspeptic attacks have become more frequent, with intervals of only a fortnight or three weeks. He attended at St. Bartholomew's Hospital for a short time and improved under treatment, but ceased to attend in consequence of a severe cough and cold followed by diarrhœa. On June 6th, 1879, he experienced a sensation

as of a heavy weight at the stomach, and it has not left him; it is worse after food.

On admission has not been sick for three weeks. Bowels act three or four times a day, motions loose. Urine sp. gr. 1026, high colour, loaded with urates. Ordered three-minim doses of Liquor Arsenicalis.

June 12th.—Bowels open four or five times.

13th.—Bowels open eight times.

14th.—Bowels open once.

16th.—Sick about an hour after the medicine. Bowels open once.

17th.—Bowels open once. Urine deep colour, sp. gr. 1035, 2·8 per cent. urea.

18th.—Urine 49 oz., urea 2·8 per cent.

19th.—Urine 36 oz., urea 2·7 per cent. On adding nitric acid to the urine there is a deep red line at the junction of the two fluids.

20th.—Has lost his appetite. Felt sick after dinner and vomited after tea. Urine not so deep in colour. Liquor Arsenicalis increased to five minims thrice a day.

22nd.—Urine 33 oz., 1·6 per cent. urea.

25th.—Liquor Arsenicalis six minims three times a day.

26th.—Urine high-coloured as usual, sp. gr. 1022; 2·2 per cent. of urea. 40 oz. of urine passed.

28th.—Urine 28 oz., 2·9 per cent. urea.

July 4th.—Liquor Arsenicalis eight minims thrice a day.

29th.—The same dose of arsenic has been taken since July 4th. The patient has improved in all respects; his bowels have become regular. He left the hospital to-day much improved.

He continued taking his medicine in eight or ten minim doses thrice a day, and he continued to improve. If he omitted his medicine he at once felt worse.

At the end of the year he was readmitted under Dr. Wilks, saying that in October vomiting came on, his appetite failed, and he began to suffer from shortness of breath. He has vomited occasionally for the last three months. His bowels have sometimes acted two or three times a day. Urine high-coloured, sp. gr. 1025.

January 7th, 1880.—Has had diarrhœa to-day.

13th.—Has had diarrhœa with much pain.

15th.—Complains of nausea.

February 2nd.—Appetite improving, and the patient feels stronger.

10th.—Complains of headache and sickness.

12th.—Still suffers from sickness.

25th.—Has severe headache and sickness, together with diarrhœa. This was stopped with starch enemas. He has been taking arsenic from time to time, but it is by no means clear that sickness and diarrhœa were due to it; and further, he has had before his admission sickness and diarrhœa when he was not taking arsenic. When he was in before, these symptoms, as well as others, improved while he was taking arsenic, and towards the end of the case he frequently had sickness when he was not taking this drug.

April 20th.—Transfusion performed by Mr. Bryant.

23rd.—Has vomited several times; great faintness.

24th.—Vomiting continues; he still suffers from faintness.

25th.—Still faint; frequent vomiting.

26th.—Vomiting continues, with occasional attacks of faintness.

27th.—Transfusion again performed by Mr. Bryant.

29th.—Has vomited two or three times.

May 1st.—Has vomited again.

5th.—Very drowsy.

12th.—Last night he had severe diarrhœa and vomiting.

13th.—Died.

Autopsy.—Fatty degeneration of the heart, œdema of the lungs. Stomach and intestines healthy. Many bones were examined, but they were perfectly normal.

Case 24. (Not published before. I have read the report in the 'Hospital Records.')—Alfred H—, æt. 52, was admitted under Dr. Wilks January 22nd, 1884. He was quite well till September, 1883, when he had an attack of diarrhœa which kept him in bed sixteen days; from this time he has gradually got weak, he has lost colour, and has developed severe symptoms of anæmia.

On admission he was suffering from constipation. The

urine was sp. gr. 1012, acid; contained some albumen, but no sugar.

February 2nd.—Suffering from constipation.

8th.—For last two days has had griping pains at the lower part of the abdomen. (He is taking arsenic.)

April 2nd.—For the last few days he has often complained of bad frontal headaches. During them he perspires freely and then breathes irregularly. For the rest of his life he often had headaches.

19th.—Urine clear, yellow, sp. gr. 1008; contains a little albumen. Soon after this date he died.

Autopsy.—There were small superficial hæmorrhages over the left temporo-sphenoidal convolutions. There was hæmorrhage round the right optic disc. There was recent pleurisy on the right side, œdema of the arytæno-epiglottidean folds. The heart was fatty, the stomach healthy. A foot above the cæcum there was a cicatrix of a small healed ulcer; it was circular, a quarter-inch in diameter, depressed with vertical margins and showed some puckering. The bones were normal.

Case 25. (Not published before. I have read the report in the 'Hospital Records.')—Edward R—, æt. 44, was admitted under Dr. Pye-Smith March 7th, 1884. Up to seven years ago he had always enjoyed good health; since then he has occasionally been out of sorts for a few days at a time, with symptoms which appear to have been dyspeptic, one of the chief of them being that he has had pain in the region of the stomach on and off for seven days at a time.

On admission.—He has abdominal pain, the bowels are constipated, and he vomits occasionally.

March 11th.—Had a slight attack of retching and vomiting; he is not taking arsenic.

April 4th.—He died.

Autopsy.—The lungs are œdematous, the heart shows fatty degeneration, there is a minute ulcer on each vocal cord. The stomach and small intestines are normal. In the colon there are a few small follicular ulcers and several healed ulcers showing pigment and puckering of the mucous membrane. There has been hæmorrhage into the mucous

membrane of the bladder. The medulla of the femur and tibia appeared to some observers normal, others considered that it was too chocolate in colour and contained too much fat.

Case 26. (Not published before. I have read the report in the 'Hospital Records.')—Clara D—, æt. 37, admitted under Dr. Pavy at the end of January, 1884. Since she was fifteen years old she has always been troubled with attacks of diarrhœa, the bowels sometimes being open seven or eight times a day. This diarrhœa is worse in the summer. Two years ago she began to suffer from sickness and abdominal pain. This attack kept her in bed some weeks; she got better, but on leaving her bed was soon taken ill again with sickness, abdominal pain, and diarrhœa. These symptoms have continued on and off till admission.

February 2nd.—She died.

On admission.—Urine sp. gr. 1015, rather dark, contains neither albumen nor bile. There are no further notes of any importance.

Autopsy.—There was over the frontal lobes a superficial hæmorrhage beneath the dura mater. There was considerable fluid in both pleuræ and in the pericardium, œdema of the lungs, fatty degeneration of the heart, fatty liver. The stomach and intestines were quite healthy.

Case 27. (Not published before. I have read the report in the 'Hospital Records.')—Marie F—, æt. 48, admitted under Dr. Pavy June 3rd, 1885. Her illness began nine months ago with weakness. Six months ago she took to her bed on account of vomiting and a burning sensation in the mouth, with epigastric pain. A few days ago she had an attack with vomiting and nausea, together with severe abdominal pain; she vomited six times in one day.

On admission.—Almost every note made of her condition up to her death on September 2nd, 1885, states that the patient was suffering from sickness, diarrhœa, and abdominal pain. No mention is made that the urine was dark. Arsenic was tried for a short time, but it was soon stopped. The sickness and diarrhœa did not bear any relationship to it.

Autopsy.—Fatty degeneration of the heart. The intestines were quite healthy. The mesenteric and other lymphatic glands, ribs, and sternum were likewise normal.

Case 28. (Not published before. I have read the report in the 'Hospital Records.')—Agnes L—, admitted under Dr. Hale White September 4th, 1886. For some time past whenever she walks about she has had palpitation and sickness.

On admission.—She is often very sick after her meals; she has no appetite. The urine is sp. gr. 1028, and contains urates. She died September 28th, 1886.

Autopsy.—The liver and heart showed fatty degeneration. The intestines, mesenteric and other lymphatic glands, and the spleen were normal. There was ante-mortem clot in the right ventricle, and embolism of the right femoral artery with gangrene of the right foot.

Case 29. (Not published before. I have read the report in the 'Hospital Records.')—Charity S—, æt. 41, admitted under Dr. Pye-Smith. She first came in December 14th, 1887, and she was discharged January 27th, 1888. She said that she had always suffered from indigestion, and often vomited directly after her food. Her urine contained lithates, with sp. gr. 1020.

January 27th.—Patient left to-day; she is very much better than when she came in. She has been taking Liquor Arsenicalis, which has gradually been pushed to ten minims thrice a day. Once only she had a little diarrhœa.

June 12th.—She was readmitted, having become very weak again, and suffering much from flatulence and sickness. The urine has a sp. gr. 1028, and gives a slight play of colour with nitric acid, but these colours do not suggest the presence of bile.

July 3rd.—Urine high colour. She has been sick occasionally.

12th.—Urine high colour. It does not contain bile.

17th.—Urine deep red colour. It contains neither bile, blood, albumen, nor sugar; sp. gr. 1020.

24th.—Urine high colour. It contains neither bile, blood, albumen, nor sugar; sp. gr. 1025.

31st.—Urine high colour. It contains neither bile, blood, albumen, nor sugar; sp. gr. 1017.

August 7th.—Urine high colour. It contains neither bile, blood, albumen, nor sugar; sp. gr. 1024.

12th.—Left to-day, better for treatment by arsenic and iron. During her stay in the hospital she has been occasionally sick.

October 29th.—Readmitted. Six weeks before readmission this time she was attacked with diarrhœa and sickness, which lasted a long while, and was followed by a return of her old illness.

On admission.—The urine was pale straw-coloured. It did not contain either bile, blood, albumen, or sugar; sp. gr. 1015. She was treated with arsenic, but she gradually sank, and died November 20th, 1888. She had no diarrhœa, and was only sick once.

Autopsy.—Heart, fatty degeneration. The liver, on treatment with ferrocyanide of potassium, showed abundance of iron. The last two feet of the small intestine showed several slate-coloured scars one third of an inch across, with loss of substance in the middle but no thickening.

An analysis by Dr. Stevenson showed that the dried liver contained 0·37 per cent. of iron. The dried spleen contained 0·184 per cent. of iron.

Case 30. (Not published before. I have read the reports in the 'Hospital Records.')—Richard W. K—, æt. 37, admitted March 11th, 1889. Two years ago he was in the hospital under Dr. Pavy for pains in the joints. The old report leaves it doubtful whether the pains were those of rheumatism or gout. His bowels have always been regular.

On admission.—Urine sp. gr. 1015, light colour, normal in all respects.

March 27th.—Vomited to-day.

April 2nd.—Had an epileptiform attack, after which he was dull for some days.

20th.—Had an attack of diarrhœa, which was stopped with Mistura Hæmatoxyli.

24th.—Urine light colour and normal.

30th.—Sank and died.

Autopsy.—Fatty degeneration of the heart. The intestines and stomach were quite healthy.

Case 31. (Not published before. I have read the reports in the 'Hospital Records.')—James L. L—, æt. 49, was admitted under Dr. Goodhart March 5th, 1889. He was in the hospital under Dr. Goodhart in 1887 for severe anæmic and retinal hæmorrhages. The urine and the state of the bowels were quite healthy. He was treated with arsenic and went out much improved.

March 5th, 1889.—Was admitted for anæmia. He improved under treatment with arsenic, and left March 13th. There was neither sickness nor diarrhœa.

June 5th.—Had again become very weak, so he was readmitted under Dr. Goodhart. He was very anæmic, and suffered from slight constipation. He was treated with arsenic. After he had been in a few days he was seized with diarrhœa and vomiting, which lasted some time. The urine was normal. He died June 30th.

Autopsy.—All the organs of the body, including the stomach and intestines, were absolutely normal.

EXPERIMENTS

ON SOME

ANTISEPTICS AND DISINFECTANTS.

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COMMUNICATED BY J. W. WASHBOURN, M.D.

THE results of experiments by numerous observers on the antiseptic value of various chemical substances differ greatly, even in the case of the antiseptics which are most commonly used in medicine and surgery. Thus Koch in his experiments ('Mittheilungen aus dem k. Gesundheitsamte,' 1881) found that corrosive sublimate, when added to peptone broth in the proportion of 1 : 300,000, completely restrained the growth in it of *Bacillus anthracis*, whilst a solution of 1 in 20,000 destroyed the vitality of the spores of anthrax in ten minutes. Klein, on the other hand (Local Government Board 'Reports,' 1885 to 1886), finds that *Bacillus anthracis* grows readily in peptone gelatin containing corrosive sublimate in the strength of 1 : 50,000, and that the spores are unaffected by a solution of 1 : 10,000 acting for one hour.

There is, therefore, need of further investigations, even with regard to such well-known substances as corrosive sublimate.

The experiments recorded in this paper have been carried out during the last twelve months in the bacteriological laboratory of Guy's Hospital under the superintendence of Dr. Washbourn.

The object of them has been to test the value of some of the more generally used antiseptics.

The substances chosen were corrosive sublimate, carbolic acid, creolin, an emulsion of coal-tar, oil of sanitas, oleum pini sylvestris.

Solutions of each of these were made with distilled water, and they were allowed to act on pure cultivations of different species of bacteria, viz.—

1. *Bacillus anthracis*, free from spores, grown in test-tubes containing 5 or 10 c.c. sterilised peptone broth.

2. The *spores* of *Bacillus anthracis* grown on the surface of potato or agar-agar.

3. *Staphylococcus pyogenes aureus*, grown in peptone broth or on the surface of agar-agar.

4. *Yellow sarcina* grown in peptone broth or on the surface of gelatin.

The points determined were—

A. The value of the substance as a disinfectant, *i. e.* its power of destroying the organisms.

B. Its value as an antiseptic, *i. e.* its power of restraining the growth of the organisms.

Methods.

A. For testing the power of killing *anthrax bacilli* possessed by each of the above-mentioned chemical substances, the following method was employed :

The anthrax bacilli were obtained in the first instance from a mouse, inoculated from an old anthrax culture which had been for some time in the laboratory. The animal died thirty-six hours after the inoculation, and a tube containing 10 c.c. peptone broth was inoculated from the blood.

From this tube all the anthrax bacilli and spores used in subsequent experiments were derived by a continuous series of cultivations, only broken from time to time by passing the organism through a mouse or a guinea-pig in order to test its virulence.

In every experiment, moreover, a control-tube of broth, gelatin, or agar was inoculated before adding the antiseptic, so as to prove the virulence and purity of each individual culture.

From one of these anthrax cultures, test-tubes, containing 5 or 10 c.c. faintly alkaline peptone broth, were inoculated by means of a platinum loop, which was always carefully dipped in right to the bottom of the tubes without touching the sides. The tubes so inoculated were placed in the incubator at 35° C. for twenty-four hours.

They were then taken out, and a drop was removed from each tube by a platinum loop and examined under the microscope.

The growth having been found to be abundant, pure, and free from spores, a control-tube of broth, gelatin, or agar was inoculated from it.

Half a c.c. to 2 c.c. of a diluted solution of the antiseptic, of known strength, was then taken up in a sterile graduated pipette, the cotton-wool plug of the test-tube was removed, and the liquid gently run in. The plug was quickly replaced, and the contents of the tube were thoroughly mixed by turning it round and round between the finger and thumb.

After the antiseptic had acted for from one minute to one hour or longer, fresh tubes of nutrient media, broth, gelatin, or agar, were inoculated from the mixture by a platinum loop. These tubes, properly labelled, were then placed in the incubator at 35° C., and examined from day to day for any growth of anthrax.

In the earlier experiments considerable difficulty was experienced in obtaining anthrax cultures which were quite free from spores.

If the bacilli were allowed to grow in a tube containing 10 c.c. peptone broth for a longer time than twenty-four hours before making the experiment, spores were frequently found on careful microscopic examination.

Almost invariably, too, when the tubes of broth were inoculated from potato cultures or from old cultures in broth or gelatin which contained spores, some of these were found remaining next day.

Ultimately I found that, in order to avoid the presence of spores, it was best, the day before the intended experiments, to inoculate the tubes containing 10 c.c. broth from broth cultures of anthrax not more than three days old.

In this way an abundant growth was obtained in twenty-

four hours, the broth became cloudy throughout, and on examination by the microscope was found to consist entirely of sporeless anthrax bacilli grouped in twos and threes.

In experimenting with the *spores of anthrax* the above method was slightly modified. Test-tubes were prepared containing small cubes of potato; these were sterilised by placing them in the steam steriliser for twenty minutes on three successive days. The surface of one or more of the cubes was then well inoculated with anthrax bacilli from a broth culture, the cotton-wool plug was cut even with the top of the tube, an india-rubber cap put on, and the tube placed in the incubator at 35° C. In five or six days the potato was covered with a thick growth of anthrax, consisting chiefly of spores. A sterile test-tube was now filled with a measured quantity of sterilised distilled water by a sterile graduated pipette. Some of the spore growth was scraped off the surface of a potato culture by a platinum loop, and was well stirred up in the water; this was repeated three or four times until the water became quite opalescent, and a drop examined under the microscope showed numerous spores in each field. A control-tube was then inoculated, and the antiseptic was added in the required quantity. After it had acted for a quarter, half, or one hour, fresh tubes of nutrient media were inoculated from the mixture, and these were placed in the incubator and examined from day to day.

As the antiseptic solutions used in these last experiments were much more concentrated than in the case of the sporeless bacilli, the inoculations were generally made with a straight platinum wire instead of a loop, so as to take up less of the fluid.

In one or two experiments the anthrax spores were taken from a growth on the surface of agar-agar instead of potato.

In the case of both the spores and the bacilli of anthrax the results obtained by these experiments were confirmed by repeating them in the same way, but after the antiseptic had acted for the required time a mouse or a guinea-pig was inoculated in addition to the tubes of nutrient media.

These last experiments, and a few others in which animals were employed, were kindly carried out for me by Dr. Washbourn.

Staphylococcus pyogenes aureus, having no spore form, was not so troublesome as *Bacillus anthracis.* In most of the experiments broth cultures one to three days old were employed. It was usually found more convenient to use smaller quantities of broth than 10 c.c. For this purpose 2, 3, or 4 c.c. were taken from the original culture by a sterile graduated pipette, and were dropped into sterile test-tubes, care being taken not to let the broth run down the sides. The antiseptic was then added, and after it had acted for certain intervals of time, fresh tubes of nutrient media were inoculated from the mixture and placed in the incubator. In a few experiments the Staphylococcus was grown on the surface of agar, and was then scraped off and stirred up in sterilised distilled water, exactly as in the case of the anthrax spores.

Experiments with *yellow sarcina* were only carried out in the case of corrosive sublimate, but the results show such extraordinary power of resistance on the part of the organism that they have seemed worthy of insertion.

The method was similar to that employed for *Staphylococcus pyogenes aureus,* but owing to the slow rate of growth of the organism, the cultures used were from ten days to four weeks old.

B. For testing the power possessed by the different chemical substances of preventing or restraining the growth of *Bacillus anthracis* and *Staphylococcus pyogenes aureus* the following was the method employed :—Test-tubes containing 5 or 10 c.c. sterilised peptone broth were mixed with $\frac{1}{2}$ to 5 c.c. of the antiseptic diluted to the required amount with sterilised distilled water. The tubes were then well inoculated with the organisms from a broth, potato, or agar culture by means of a platinum loop, and were placed in the incubator at 35° C. Each day they were examined to see if any growth had occurred. In every case a tube of broth, gelatin, or agar was inoculated as a control.

At the end of this paper will be found the experiments on the action of *ipecacuanha* on anthrax bacilli free from spores, which are referred to in Mr. Davies-Colley's article in this volume (p. 1). The bacilli used for these experiments were grown in test-tubes containing 5 c.c. peptone broth. Twenty-

four hours after inoculation a drop was examined under the microscope and a control-tube was inoculated. A weighed quantity of powdered ipecacuanha root was then taken up on a sterile copper spatula and dropped into the tube. The contents were gently shaken up, and, after the ipecacuanha had acted for a quarter, half, or one hour, fresh tubes of nutrient media were inoculated from the mixture. These were placed in the incubator, and examined from day to day.

In many of these tubes there occurred an abundant growth of motile and non-motile bacilli, which could easily be distinguished from anthrax bacilli, both by their mode of growth and by the microscope. They were evidently introduced along with the ipecacuanha, as this was not sterilised in any way before use.

As it seemed possible that instead of having been killed by the ipecacuanha, the anthrax bacilli might in some cases be merely prevented from growing owing to the presence of other organisms, fresh tubes of nutrient media were in several cases inoculated simultaneously with these impure growths and with actively growing anthrax bacilli. The latter continued to grow for the first forty-eight hours, but after that time they gradually disappeared, being apparently destroyed by the other bacilli.

To eliminate more completely still this source of fallacy, three experiments were performed in the following manner:

A tube containing 5 c.c. peptone broth was inoculated with anthrax bacilli and was placed in the incubator. After twenty-four hours it was taken out, and a drop of the broth was examined under the microscope to ascertain the purity of the growth. A weighed quantity of ipecacuanha was then taken up on a copper spatula and was dropped into the tube; the contents were well shaken up and, after the powder had acted for a quarter and half hour, test-tubes containing about 10 c.c. peptone gelatin were inoculated from the mixture by a platinum loop.

The gelatin in these tubes was then melted by gently warming, and the contents of each tube were poured out on to a sterile glass plate, placed in a damp chamber. In this way the different organisms which had been introduced into the gelatin were separated from one another, and grew

independently in distinct colonies which could be examined under the microscope, and thus any growth of anthrax could be at once ascertained.

EXPERIMENTS.

A. To Determine the Strength of the Antiseptic necessary to Destroy the Micro-organisms.

Corrosive Sublimate.

A stock solution of this substance, strength 1 : 50, was made by dissolving 5 grammes of the pure salt in 250 c.c. distilled water. This was kept in a well-stoppered bottle, and was renewed about every two months. The more dilute solutions were made fresh for each experiment, and the following precautions were taken to prevent them from becoming contaminated by organisms derived from the air or elsewhere :—A measured quantity of the stock solution was taken up in a sterile graduated pipette, and was run into a sterile flask, the mouth of which was plugged with cotton wool. Sterilised distilled water was then added in the required amount, and the contents of the flask were very thoroughly mixed. By means of a sterile pipette, graduated accurately in tenths of a cubic centimetre, $\frac{1}{2}$ to 2 c.c. were removed from the flask, and gently run into the test-tubes containing the organisms which were to be acted on by the antiseptic solution.

An example will perhaps show best the kind of calculation required in making up the dilute solutions. Thus, in the experiments made to determine the action of a solution of corrosive sublimate, strength 1 : 25,000, on anthrax bacilli growing in peptone broth, 5 c.c. of the stock solution (1 : 50) were placed in a litre flask. To this were added 245 c.c. sterilised distilled water, giving a solution 1:2500; $1\frac{1}{10}$ c.c. of this added to the 10 c.c. peptone broth containing the anthrax bacilli, gave a solution as nearly as possible of the required strength 1 : 25,000. The exact proportion is 1 : 25,227 and a fraction, but the error involved in taking the round numbers is very slight, and is, moreover, compensated by any evaporation of

the 10 c.c. broth during the twenty-four hours spent in the incubator at 35° C.

I. *Action on Sporeless Anthrax Bacilli.*

1. Several preliminary experiments showed that the vitality of the bacilli was unaffected by corrosive sublimate solutions of strength 1 : 60,000, 1 : 50,000, 1 : 40,000, acting for a quarter, half, and one hour.

2. 1 : 30,000.

A test-tube containing 10 c.c. peptone broth was inoculated with anthrax bacilli. It was placed in the incubator at 35° C. for twenty-four hours. A control-tube was then inoculated from it, and corrosive sublimate was added in such quantity as to form a solution of the above strength. From this mixture a tube of peptone broth and one of agar were inoculated after a quarter of an hour, a tube of agar after half an hour, a tube of broth and one of agar after one hour. All these tubes showed next day an abundant and pure growth of anthrax bacilli.

3. 1 : 25,000.

In two experiments the corrosive sublimate was added to 10 c.c. peptone broth containing a twenty-four hours' growth of anthrax, so as to form a solution 1 : 25,000.

Exp. A.—From one tube containing this mixture, two tubes of broth were inoculated after a quarter of an hour, and a tube of agar after half an hour. Next day there was a good growth of anthrax bacilli in one of the tubes of broth, but the other two tubes remained free from growth of any kind.

Exp. B.—From a second tube containing a similar mixture, a tube of broth was inoculated after one minute, and a tube of gelatin after a quarter of an hour. Both of these tubes showed next day a good growth of anthrax bacilli. A tube of broth also inoculated after a quarter of an hour, and another tube of broth inoculated after half an hour, remained free from growth.

4. 1 : 20,000.

In two experiments the corrosive sublimate was added in the above proportion to 10 c.c. broth, containing a twenty-four hours' growth of anthrax.

Exp. A.—From a tube containing this mixture, a tube of broth was inoculated after a quarter of an hour, and next day showed a good growth of anthrax. A tube of broth inoculated after half an hour, a tube of broth and one of gelatin inoculated after one hour, remained free from growth.

Exp. B.—From a second tube, two tubes of broth were inoculated after one minute and a quarter of an hour respectively, and next day showed a good growth of anthrax. A tube of broth and one of gelatin inoculated after half an hour remained free from growth.

5. 1 : 15,000.

In four experiments the corrosive sublimate was added in the above proportion to 10 c.c. broth containing a twenty-four hours' growth of anthrax.

Exp. A.—From the first tube containing this mixture, six tubes of broth were inoculated in pairs after a quarter, half, and one hour respectively. They all remained free from growth.

Exp. B.—From a second tube, three tubes of broth were inoculated after one, two, and three minutes respectively, a tube of broth and one of agar were inoculated after five minutes, a tube of broth and one of agar after ten minutes, two tubes of broth and one of agar after a quarter of an hour, two tubes of broth after half an hour. These all remained free from any kind of growth.

Exp. C.—From a third tube, before adding the antiseptic, a mouse was inoculated as a control. The sublimate was then added in the above proportion. A mouse was inoculated from the mixture after a quarter of an hour, and another after half an hour. Both these latter animals remained alive and well; the control mouse died in thirty-six hours, and its blood was found to be swarming with anthrax bacilli.

Exp. D.—From a fourth tube, a control mouse was first inoculated. The sublimate was then added, and after a quarter of an hour a mouse was inoculated from the mixture. The control mouse died of anthrax within forty-eight hours, the other one remained alive and well.

Conclusions.—From these experiments it appears that anthrax bacilli growing in 10 c.c. peptone broth are destroyed by corrosive sublimate solutions—

1 : 15,000 acting for one minute,
1 : 20,000 and 1 : 25,000 acting for half an hour;
but they are unaffected by solutions—
1 : 20,000 and 1 : 25,000 acting for a quarter of an hour,
1 : 30,000 acting for one hour.

II. *Action on Anthrax Spores.*

1. Preliminary experiments showed that corrosive sublimate solutions of strength 1 : 10,000 and 1 : 5000, acting for a quarter, a half, and one hour, had no effect on the vitality of the spores of anthrax.

2. 1 : 3000.

In two experiments sterile test-tubes were filled with 5 c.c. sterilised distilled water. This was well inoculated with anthrax spores taken from the surface of potato. The sublimate was then added in the above proportion.

Exp. A.—From one of these tubes, a tube of agar was inoculated after five minutes, and two tubes of broth after a quarter and half an hour respectively. Next day these tubes all showed a good growth of anthrax. Two tubes of agar inoculated after one hour remained free from growth.

Exp. B.—From another tube, a tube of broth was inoculated after ten minutes, and a tube of broth and one of agar after half an hour. These all showed a good growth of anthrax next day.

3. 1 : 1500.

In two experiments corrosive sublimate was added to 5 c.c. sterilised distilled water which had previously been well inoculated with anthrax spores from a growth on potato.

Exp. A.—From one of the tubes containing this mixture, a tube of agar was inoculated after two minutes, and three tubes of broth after five minutes, a quarter, and half an hour respectively. These all showed a good growth of anthrax in twenty-four hours. A tube of broth and one of agar were inoculated after one hour, and they remained free from growth.

Exp. B.—From another tube containing a similar mixture, a tube of broth was inoculated after a quarter of an hour, and showed good growth of anthrax next day. A tube of gelatin inoculated after half an hour, a tube of broth and one of

gelatin inoculated after one hour, and a tube of gelatin inoculated after four hours, all remained free from growth.

4. 1 : 1000.

In four experiments corrosive sublimate was added to 4 c.c. sterilised distilled water in which anthrax spores were suspended. In Exp. A the spores were taken from a growth on the surface of agar; in Exps. B, C, D they were taken from potato.

Exp. A.—From the first tube, two tubes of broth were inoculated after one and two minutes respectively. These showed good growth of anthrax next day. A tube of broth inoculated after five minutes, and another inoculated after a quarter of an hour, remained free from growth.

Exp. B.—From a second tube, three tubes of broth were inoculated after one, two, and five minutes respectively. These next day showed a good growth of anthrax. A tube of broth and one of agar inoculated after a quarter of an hour remained free from growth.

Exp. C.—From a third tube, a tube of agar was inoculated after a quarter of an hour; in three days' time a single small spot of growth appeared at the top of the sloping surface of the agar; this was found on microscopical examination to consist of anthrax bacilli and spores. A tube of broth and one of gelatin inoculated after half an hour, a tube of broth and one of agar inoculated after one hour, all remained free from growth.

Exp. D.—From a fourth tube, before adding the antiseptic, a guinea-pig was inoculated as a control. The corrosive sublimate was then added in the proportion 1 : 1000, and after a quarter of an hour a second guinea-pig was inoculated. The control animal died in forty-eight hours, and its blood was found to be full of anthrax bacilli; the other one remained alive and well.

Conclusions.—These experiments show that anthrax spores are destroyed by corrosive sublimate solutions—

1 : 1000 acting for a quarter of an hour,
1 : 1500 and 1 : 3000 acting for one hour;

but they are unaffected by solutions—

1 : 1000 acting for five minutes,
1 : 1500 acting for a quarter of an hour,
1 : 3000 acting for half an hour,
1 : 5000 acting for one hour.

III. *Action on Staphylococcus pyogenes aureus.*

1. 1 : 3000.

Corrosive sublimate was added in the above proportion to 5 c.c. broth, which contained a two days' growth of *Staphylococcus aureus*. From this mixture a tube of broth and one of agar were inoculated after a quarter of an hour, a tube of gelatin after half an hour, a tube of broth and one of agar after one hour, and a tube of broth after twenty-four hours.

In two days there was an abundant and pure growth of *Staphylococcus aureus* in all the tubes.

2. 1 : 1000.

Exp. A.—The corrosive sublimate was added in the above proportion to 10 c.c. broth containing a growth of *Staphylococcus aureus* three days old. From this mixture a tube of broth and one of gelatin were inoculated after a quarter of an hour, a tube of broth and one of agar after half an hour, a tube of broth and one of agar after one hour, and a tube of broth after twenty-four hours. In two days all the tubes showed an active growth of *Staphylococcus aureus*.

Exp. B.—The corrosive sublimate was added to 10 c.c. broth containing a growth of *Staphylococcus aureus* one day old. A tube of broth and one of agar inoculated from this mixture after half an hour, showed an active growth in two days.

3. 1 : 600.

The corrosive sublimate was added to 5 c.c. broth containing a growth of *Staphylococcus aureus* three days old. A tube of broth and one of gelatin were inoculated from the mixture after a quarter of an hour, a tube of broth and one of agar after half an hour, and a tube of agar after one hour. These tubes all showed normal growth of *Staphylococcus aureus* in two days. A tube of agar inoculated after twenty-four hours showed no signs of growth for seven days, but then several minute colonies of Staphylococci appeared, and spread rapidly.

4. 1 : 500.

Exp. A.—The corrosive sublimate was added to 4½ c.c. broth containing a growth of *Staphylococcus aureus* one day old. Two tubes of agar inoculated from this mixture after a quarter and half an hour respectively, and a tube of gelatin inoculated

after an hour, showed good growth of *Staphylococcus aureus* in two days. A tube of agar inoculated after four hours remained free from growth.

Exp. B.—A sterile test-tube was filled with 4½ c.c. sterilised distilled water; this was then well inoculated with *Staphylococcus aureus* taken from a culture on the surface of agar-agar, and corrosive sublimate was added in the above proportion. From this mixture a tube of agar and one of gelatin were inoculated after a quarter of an hour, a tube of agar after half an hour, and a tube of broth and one of agar after an hour. In all these tubes there was a normal growth of *Staphylococcus aureus* within two days. A tube of agar and one of gelatin inoculated after twenty-four hours, showed no sign of growth for six days, but then several small colonies of *Staphylococcus aureus* appeared in each.

5. 1 : 250.

Exp. A.—The corrosive sublimate was added to 5 c.c. broth containing a growth of *Staphylococcus aureus* three days old. A tube of broth and one of agar inoculated from the mixture after a quarter of an hour, and a tube of broth inoculated after half an hour, showed normal growth of *Staphylococcus aureus* in two days. A tube of broth and one of agar inoculated after an hour showed a rather scanty growth in three days; a tube of agar inoculated after twenty-four hours showed a very scanty growth after five days.

Exp. B.—The corrosive sublimate was added to 5 c.c. broth containing a growth two days old. A tube of agar and one of gelatin inoculated from the mixture after a quarter of an hour showed normal growth of *Staphylococcus aureus* in two days. A tube of agar inoculated after half an hour, and a tube of broth inoculated after an hour, remained free from growth for six days, but then a few small colonies of Staphylococci appeared. A tube of broth inoculated after half an hour and a tube of agar inoculated after an hour remained free from growth.

6. 1 : 200.

Exp. A.—Corrosive sublimate was added in the above proportion to 3 c.c. broth containing a growth of *Staphylococcus aureus* two days old. Five tubes of agar inoculated from this mixture after a quarter, half, one, four, and five hours respectively, remained free from growth of any kind.

Exp. B.—A sterile test-tube was filled with 3 c.c. sterilised distilled water; this was well inoculated with *Staphylococcus aureus* from a growth on the surface of agar, and corrosive sublimate was added in the above proportion. From this mixture a tube of agar was inoculated after five minutes, a tube of gelatin after a quarter of an hour, a tube of agar and one of gelatin after half an hour, and a tube of gelatin after one hour. All the tubes remained free from any kind of growth.

7. 1 : 150.

Corrosive sublimate was added in the above proportion to 2 c.c. broth containing a growth of *Staphylococcus aureus* two days old. A tube of broth and one of agar inoculated from this mixture after a quarter of an hour, and a tube of broth and one of agar inoculated after half an hour, all remained free from growth.

Conclusions.—From these experiments it appears that the Staphylococci, whether growing in broth or suspended in distilled water, are destroyed by corrosive sublimate solutions—

1 : 200 and 1 : 150 acting for a quarter of an hour.

The vitality of the organism is, however, markedly impaired by solutions—

1 : 600 and 1 : 500 acting for twenty-four hours,
1 : 250 acting for one hour;

whilst they are unaffected by solutions—

1 : 1000 acting for twenty-four hours,
1 : 600 and 1 : 500 acting for one hour,
1 : 250 acting for half an hour.

IV. *Action on Yellow Sarcina.*

1. Several preliminary experiments showed that the vitality of yellow sarcina was not affected by corrosive sublimate in the proportions of 1 : 20,000, 1 : 10,000, 1 : 5000, 1 : 2000, acting for half and one hour.

2. 1 : 1000.

Corrosive sublimate was added in the above proportion to 10 c.c. broth containing a pure growth of yellow sarcina ten days old.

From this mixture three tubes of broth were inoculated after five minutes, a quarter, and one hour respectively.

In five days' time there was an abundant growth of yellow sarcina in all the tubes, quite equal to that in the control-tube.

3. 1 : 500.

A sterile test-tube was filled with 10 c.c. sterilised distilled water; this was well inoculated with yellow sarcina taken from a culture on a sloping surface of gelatin. A control-tube was then inoculated, and the corrosive sublimate added in the above proportion.

A tube of gelatin inoculated from the mixture after five minutes, and another inoculated after a quarter of an hour, showed a normal amount of growth in four days. A tube of agar and one of gelatin inoculated after half an hour showed a fair amount of growth in six days. A tube of broth inoculated after twenty-four hours remained free from growth.

4. 1 : 300.

Corrosive sublimate was added in the above proportion to 5 c.c. broth containing a growth of yellow sarcina two weeks old.

A tube of broth inoculated from the mixture after five minutes showed a normal growth of yellow sarcina in five days. A tube of broth and one of agar inoculated after a quarter of an hour showed a rather scanty growth in six days. A tube of gelatin inoculated after half an hour, and a tube of agar inoculated after one hour, showed several small colonies of growth in ten days. A tube of agar inoculated after twenty-four hours remained free from growth for twelve days, but then a few small spots appeared and spread slowly.

5. 1 : 100.

Exp. A.—Corrosive sublimate was added in the above proportion to 5 c.c. broth containing a growth of yellow sarcina four weeks old. A tube of gelatin inoculated from this mixture after five minutes showed no sign of growth for ten days, but then several colonies appeared and slowly spread.

Another tube of gelatin inoculated after a quarter of an hour showed after three weeks a few scattered points of growth. A tube of broth also inoculated after a quarter of an hour, a tube of broth and one of agar inoculated after half

an hour, and a tube of broth and one of gelatin inoculated after an hour, remained free from growth.

Exp. B.—A sterile test-tube was filled with 5 c.c. sterilised distilled water; this was well inoculated with yellow sarcina from a culture on gelatin, and then corrosive sublimate was added in the above proportion.

Four tubes of gelatin inoculated from this mixture after five minutes, a quarter of an hour, half an hour, and one hour respectively, remained free from growth.

Conclusions.—These experiments show that yellow sarcina is destroyed by corrosive sublimate solutions—

1 : 100 acting for half an hour.

The vitality of the organisms is, however, almost destroyed by solutions—

1 : 100 acting for a quarter of an hour;

and is distinctly impaired by solutions—

1 : 300 acting for a quarter of an hour,

1 : 500 acting for half an hour.

The organisms are unaffected by solutions—

1 : 500 acting for a quarter of an hour,

1 : 1000 acting for an hour.

Carbolic Acid.

A stock solution of this substance, strength 1 : 20, was made with distilled water, and was kept in a stoppered bottle. The more dilute solutions were made fresh for each experiment, with the same precautions as in the case of corrosive sublimate.

I. *Action on Sporeless Anthrax Bacilli.*

1. 1 : 300.

The carbolic acid was added to 10 c.c. broth containing a twenty-four hours' growth of anthrax bacilli in such proportion as to form a solution of the above strength. From a test-tube containing this mixture a tube of agar was inoculated after one minute, a tube of broth and one of agar after a quarter of an hour, a tube of broth and one of gelatin after half an hour, a tube of agar after one hour. Next day all these tubes showed a normal growth of anthrax.

2. 1 : 175.

In two experiments carbolic acid was added in the above proportion to 5 c.c. broth, containing a twenty-four hours' growth of anthrax bacilli.

Exp. A.—From a tube containing this mixture a tube of broth was inoculated after one minute, and next day showed a normal growth of anthrax. Two tubes of gelatin and one of agar inoculated after half an hour remained free from growth.

Exp. B.—From a second tube containing a similar mixture a tube of gelatin was inoculated after one minute, and a tube of agar and one of gelatin after a quarter of an hour. All these tubes showed a normal growth of anthrax next day.

3. 1 : 150.

Carbolic acid was added in the above proportion to 5 c.c. broth containing a twenty-four hours' growth of anthrax bacilli. A tube of broth was inoculated from the mixture after one minute, and showed a normal growth of anthrax next day. Another tube of broth was inoculated after a quarter of an hour, a tube of broth and one of gelatin after half an hour, a tube of broth and one of agar after one hour. These all remained free from growth.

4. 1 : 100.

In two experiments carbolic acid was added in the above proportion to 10 c.c. broth, containing a twenty-four hours' growth of anthrax bacilli.

Exp. A.—From a tube containing this mixture a tube of broth was inoculated after five minutes and a tube of agar after a quarter of an hour. Both tubes remained free from growth.

Exp. B.—From a second tube containing a similar mixture a tube of agar was inoculated after a quarter of an hour, a tube of broth after half an hour, a tube of agar and one of gelatin after one hour. All the tubes remained free from growth.

Conclusions.—These experiments show that anthrax bacilli free from spores are destroyed by carbolic acid solutions—

1 : 100 acting for five minutes,
1 : 150 acting for a quarter of an hour,
1 : 175 acting for half an hour;

but they are unaffected by solutions—

1 : 150 acting for one minute,
1 : 175 acting for a quarter of an hour,
1 : 300 acting for one hour.

II. *Action on Anthrax Spores.*

1. 1 : 100.

A sterile test-tube was filled with 5 c.c. sterilised distilled water; this was well inoculated with anthrax spores from a culture on potato, and carbolic acid was added in the above proportion. A tube of agar was inoculated from the mixture after a quarter of an hour, a tube of broth after half an hour, a tube of agar after one hour, and a tube of broth after three hours. Next day all the tubes showed a pure and normal growth of anthrax.

2. 1 : 20.

5 c.c. of the stock solution of carbolic acid (1 : 20) were put into a sterile test-tube. The liquid was then well inoculated with anthrax spores from a culture on potato. A tube of broth was inoculated from the mixture after a quarter of an hour, a tube of gelatin after half an hour, a tube of broth and one of gelatin after one hour, and a tube of gelatin after four hours. In all these tubes there occurred a normal growth of anthrax. A tube of broth and one of gelatin inoculated after twenty-four hours remained free from growth.

Conclusions.—From these experiments it appears that anthrax spores are killed by a carbolic solution—

1 : 20 acting for twenty-four hours,

but that they are unaffected by the same solution acting for half, one, or four hours.

III. *Action on Staphylococcus pyogenes aureus.*

1. 1 : 50.

Carbolic acid was added in the above proportion to 4 c.c. broth, containing a growth of *Staphylococcus pyogenes aureus* two days old. From this mixture a tube of agar was inoculated after a quarter of an hour, a tube of broth after half an

hour, a tube of agar and one of gelatin after one hour. All the tubes remained free from growth.

2. 1 : 40.

Carbolic acid was added in the above proportion to 2 c.c. broth containing a growth of *Staphylococcus aureus* one day old. From this mixture a tube of agar was inoculated after a quarter of an hour, a tube of broth after half an hour, a tube of broth and one of agar after one hour. All the tubes remained free from growth.

Conclusions.—These two experiments show that carbolic acid solutions 1 : 50 and 1 : 40 completely kill all the Staphylococci contained in a broth culture after acting for a quarter of an hour.

Creolin.

The dilute solutions, as required for each experiment, were made with sterilised distilled water, the same precautions being observed as in the case of corrosive sublimate.

I. *Action on Sporeless Anthrax Bacilli.*

1. 1 : 500.

A solution of creolin was added in the above proportion to 10 c.c. broth, containing a twenty-four hours' growth of anthrax bacilli. From the test-tube containing this mixture five tubes of broth and five tubes of agar were inoculated, two at a time, after one minute, a quarter, a half, one, and four hours. The next day there was an active growth of anthrax bacilli in all the tubes except two tubes of agar inoculated after a quarter and a half hour respectively. These remained free from growth.

2. 1 : 400.

Creolin was added in the above proportion to 10 c.c. broth, containing a twenty-four hours' growth of anthrax bacilli. From this mixture two tubes of broth were inoculated after one minute and a quarter of an hour respectively, a tube of broth and one of agar after half an hour, a tube of broth and one of gelatin after one hour. All the tubes next day showed

a good growth of anthrax, except the tube of broth inoculated in a quarter of an hour, which remained free from growth.

3. 1 : 300.

Creolin was added in the above proportion to 10 c.c. broth, containing a twenty-four hours' growth of anthrax bacilli. A tube of agar inoculated from the mixture after one minute, a tube of broth and one of gelatin inoculated after a quarter of an hour, a tube of broth and one of agar inoculated after half an hour, all remained free from growth.

4. 1 : 200.

Creolin was added in the above proportion to 10 c.c. broth, containing a twenty-four hours' growth of anthrax bacilli. A tube of agar was inoculated from this mixture after one minute, a tube of agar and one of gelatin after a quarter of an hour, a tube of agar and one of gelatin after half an hour. All these tubes remained free from growth.

5. 1 : 150.

Creolin was added in the above proportion to 5 c.c. broth, containing a twenty-four hours' growth of anthrax bacilli. A tube of broth inoculated from the mixture after one minute, and two tubes of agar inoculated after a quarter of an hour, remained free from growth.

Conclusions.—From these experiments it appears that spore-less anthrax bacilli are destroyed by creolin in the proportions

1 : 300 and 1 : 200 after one minute,

but that they are unaffected by a solution—

1 : 400 acting for one hour.

II. *Action on Anthrax Spores.*

1. 1 : 30.

Creolin was added in the above proportion to 5 c.c. sterilised distilled water, which had previously been well inoculated with anthrax spores from a potato culture. From this mixture four tubes of broth and four tubes of agar were inoculated, two at a time, after a quarter of an hour, two days, four days, and six days respectively. All the tubes showed a good growth of anthrax bacilli in twenty-four hours.

In this and subsequent experiments it was found that the creolin which had been added to the water containing the

anthrax spores, underwent decomposition after twenty-four hours. A black tarry precipitate then gradually fell to the bottom of the test-tube, leaving the supernatant fluid somewhat turbid.

2. 1 : 10.

Exp. A.—Creolin was added in the above proportion to 4½ c.c. sterilised distilled water, which had been previously well inoculated with anthrax spores from a culture on potato. From this mixture a tube of broth was inoculated after five minutes, a tube of broth and one of agar after a quarter of an hour, a tube of broth and one of agar after one hour, a tube of broth after five hours, a tube of broth and one of gelatin after two days. Good growth of anthrax occurred in all except the tube of agar inoculated in a quarter of an hour, which remained free from growth.

Exp. B.—A sterile test-tube was filled with 4½ c.c. sterilised distilled water. This was well inoculated with anthrax spores from a culture on the surface of agar, and a mouse was inoculated from the mixture as a control. The creolin was then added in the proportion 1 : 10, and after half an hour a guinea-pig was inoculated from the mixture. From the same mixture a tube of broth was inoculated after a quarter of an hour, and a tube of gelatin after three quarters of an hour. Both these tubes showed a good growth of anthrax bacilli after two days, but the guinea-pig remained alive and well. The mouse died in forty-eight hours, and its blood was found to be swarming with anthrax bacilli.

Exp. C.—A test-tube was filled with 4½ c.c. sterilised distilled water, and this was well inoculated with anthrax spores from a potato culture. A mouse was inoculated from the contents of the tube as a control, and creolin was then added in the proportion 1 : 10. After half an hour a guinea-pig was inoculated from the mixture, and immediately after this a tube of broth and one of agar were also inoculated. These tubes both showed a good growth of anthrax bacilli next day, but the guinea-pig remained alive and well. The mouse died in forty-eight hours, and its blood was found to be full of anthrax bacilli.

Conclusions.—From these experiments it appears that anthrax spores, after remaining in a solution of creolin 1 : 10

for as long as forty-eight hours, are still able to grow when transplanted into a suitable medium such as peptone broth. The virulence of the spores, however, seems to have become weakened or destroyed at a much earlier period, since two guinea-pigs, inoculated with the spores after the creolin had only been acting for half an hour, remained alive and unaffected.

III. *Action on Staphylococcus pyogenes aureus.*

1. 1 : 100.

Creolin was added in the above proportion to 5 c.c. broth containing a growth of *Staphylococcus aureus* three days old. From this mixture a tube of broth and one of agar were inoculated after a quarter of an hour, a tube of gelatin after half an hour, a tube of broth and one of agar after one hour. In two days' time all these tubes showed an active growth of *Staphylococcus aureus.* A tube of agar inoculated after twenty-four hours, and a tube of broth and one of agar inoculated after three days, remained free from growth.

2. 1 : 75.

Creolin was added in the above proportion to 4 c.c. broth, containing a growth of *Staphylococcus aureus* two days old. From this mixture a tube of broth and one of agar were inoculated after a quarter of an hour, a tube of agar after half an hour, and a tube of broth after one hour. No growth of *Staphylococcus aureus* occurred in any of the tubes, but after four days a long thick bacillus was found to be growing in one of the tubes of broth; probably it had been accidentally introduced when the tube was inoculated.

3. 1 : 50.

Creolin was added in the above proportion to 5 c.c. broth, containing a growth of *Staphylococcus aureus* two days old. From this mixture a tube of broth was inoculated after a quarter of an hour, a tube of gelatin after half an hour, a tube of broth and one of agar after one hour. All these tubes remained free from growth.

Conclusions.—These experiments show that all the Staphylococci in a broth culture are destroyed by creolin in the proportions—

1 : 100 acting for twenty-four hours,
1 : 75 acting for a quarter of an hour;
but that the organisms are unaffected by a solution—
1 : 100 acting for one hour.

Coal-tar Emulsion (Liquor Picis Carbonis).

This emulsion was made for me by Mr. Collier, dispenser to the hospital, from a formula published by him in the 'Pharmaceutical Journal,' March 15th, 1890.

It corresponds to the Liquor Picis Carbonis of the B.P. unofficial formulary, but the coal-tar is emulsified by resin soap instead of infusion of quillaia.

The mode of preparation is as follows:

Take of resin soap 2 oz.,
Rectified spirit 1 pint;
dissolve the soap in the spirit by means of a gentle heat, and then add—
Prepared coal-tar 4 oz.

Digest at a temperature of 120° F. for two days, allow to become cold, and filter. The product is a dark, thick liquid, resembling creolin. When mixed with water, it forms an opaque emulsion, which does not separate on standing.

As in the case of creolin, dilute solutions of this substance were made with sterilised distilled water, in the proportions required for each experiment.

I. *Action on Sporeless Anthrax Bacilli.*

1. 1 : 500.

The coal-tar emulsion was added in the above proportion to 10 c.c. broth containing a twenty-four hours' growth of anthrax bacilli.

From this mixture five tubes of broth and five of agar were inoculated, two at a time, after one minute, a quarter, half, one, and four hours respectively. Next day all the tubes showed an active growth of anthrax, except two tubes of agar, inoculated after a quarter and half an hour respectively; these remained free from growth.

2. 1 : 400.

The emulsion was added in the above proportion to 10 c.c. broth containing a twenty-four hours' growth of anthrax bacilli.

From this mixture two tubes of broth were inoculated after one minute and a quarter of an hour respectively, a tube of broth and one of gelatin were inoculated after half an hour, a tube of broth and one of agar after one hour.

A good growth of anthrax occurred next day in the tube of broth inoculated in one minute, and in five days a scanty growth of anthrax appeared in the tube of gelatin inoculated in half an hour. The other tubes all remained free from growth.

3. 1 : 300.

The emulsion was added in the above proportion to 10 c.c. broth containing a twenty-four hours' growth of anthrax bacilli.

A tube of agar inoculated from the mixture after one minute, a tube of broth and one of gelatin inoculated after a quarter of an hour, and a tube of broth and one of agar inoculated after half an hour, all remained free from growth.

4. 1 : 200.

The emulsion was added to 10 c.c. broth containing a twenty-four hours' growth of anthrax bacilli.

A tube of gelatin inoculated from the mixture after one minute, a tube of agar and one of gelatin inoculated after a quarter of an hour, and a tube of agar and one of gelatin inoculated after half an hour, all remained free from growth.

Conclusions.—These experiments show that anthrax bacilli growing in peptone broth are destroyed by coal-tar emulsion in the proportions—

1 : 400 after one hour,
1 : 300 and 1 : 200 after one minute;

whilst they are unaffected by the emulsion in the proportions

1 : 400 after one minute,
1 : 500 after four hours.

II. *Action on Anthrax Spores.*

1. 1 : 30.

The coal-tar emulsion was added in the above proportion to 5 c.c. sterilised distilled water which had previously been well inoculated with anthrax spores from a culture on potato.

From this mixture four tubes of broth and four tubes of agar were inoculated after a quarter of an hour, two days, four days, and six days respectively. All the tubes showed a good growth of anthrax in twenty-four hours.

2. 1 : 10.

In two experiments the emulsion was added in the above proportion to test-tubes containing 4½ c.c. sterilised distilled water which had been well inoculated with anthrax spores from a culture on potato.

Exp. A.—From one tube containing this mixture a tube of broth was inoculated after five minutes, a tube of broth and one of agar after a quarter of an hour, a tube of broth and one of agar after half an hour, a tube of broth after five hours, a tube of broth and one of gelatin after two days.

Good growth of anthrax occurred in all these tubes, except the tube of agar inoculated after one hour and the tube of gelatin inoculated after forty-eight hours, which both remained free from growth.

Exp. B.—From another tube containing a similar mixture a tube of broth was inoculated after a quarter of an hour, a tube of gelatin after half an hour, a tube of broth and one of gelatin after one hour, a tube of gelatin after four hours, and another tube of gelatin after twenty-four hours.

All these tubes showed a good growth of anthrax in twenty-four hours.

Conclusions.—From these experiments it appears that anthrax spores are not killed by coal-tar emulsion in the proportion—

1 : 10 acting for two days.

III. *Action on Staphylococcus pyogenes aureus.*

1. 1 : 100.

Coal-tar emulsion was added in the above proportion to 2 c.c. broth containing a growth of *Staphylococcus aureus*

two days old. From the test-tube containing this mixture a tube of broth and one of agar were inoculated after a quarter of an hour, a tube of agar after half an hour, and a tube of broth and one of agar after one hour. In all these tubes there occurred a normal growth of *Staphylococcus aureus.*

2. 1 : 75.

The emulsion was added in the above proportion to 4 c.c. broth containing a growth of *Staphylococcus aureus* two days old. From this mixture a tube of agar was inoculated after a quarter of an hour, a tube of broth and one of agar after half an hour, a tube of agar after three hours, and three more tubes of agar after five, twenty-four, and forty-eight hours respectively. All these tubes showed a good growth of *Staphylococcus aureus* within forty-eight hours.

3. 1 : 40.

The emulsion was added in the above proportion to 2 c.c. broth containing a one day's growth of *Staphylococcus aureus.* From this mixture a tube of agar was inoculated after a quarter of an hour, a tube of broth after half an hour, a tube of agar after one hour, a tube of broth and one of agar after four hours, and a tube of broth after twenty-four hours. All these tubes showed a normal growth of *Staphylococcus aureus.*

Conclusions.—These experiments show that the coal-tar emulsion, while slightly superior to creolin in its power of destroying anthrax bacilli, is markedly inferior to it in its action on *Staphylococcus pyogenes aureus.* This organism growing in broth was unaffected by the emulsion in the proportions—

1 : 75 acting for forty-eight hours,
1 : 40 acting for twenty-four hours.

Oil of Sanitas.

In order to render this substance miscible with water or broth, it was emulsified by adding to it a sufficient quantity of resin soap.

Experiments made with the resin soap alone showed that

this had no action either upon anthrax bacilli or on *Staphylococcus pyogenes aureus.*

I. *Action on Sporeless Anthrax Bacilli.*

1. 1 : 500.

The emulsified oil of sanitas was added in the above proportion to 10 c.c. broth containing a twenty-four hours' growth of anthrax bacilli.

Four tubes of broth inoculated from the mixture after one minute, five minutes, quarter of an hour, and half an hour respectively, all showed good growth of anthrax next day.

2. 1 : 250.

Oil of sanitas was added in the above proportion to 5 c.c. broth containing a twenty-four hours' growth of anthrax bacilli. Four tubes of broth inoculated from this mixture after five minutes, a quarter of an hour, half an hour, and one hour respectively, all showed normal growth of anthrax next day.

3. 1 : 200.

Oil of sanitas was added in the above proportion to 10 c.c. broth containing a twenty-four hours' growth of anthrax bacilli. From this mixture a tube of broth and one of gelatin were inoculated after a quarter of an hour, a tube of broth and one of agar after half an hour, and a tube of gelatin after one hour. All these tubes remained free from growth.

4. 1 : 150.

Oil of sanitas was added in the above proportion to 5 c.c. broth containing a twenty-four hours' growth of anthrax bacilli. A tube of broth and one of agar inoculated from this mixture after one minute, both showed a good growth of anthrax next day. A tube of broth and one of agar inoculated after a quarter of an hour, and a tube of broth and one of gelatin inoculated after one hour, remained free from growth.

Conclusions.—These experiments show that anthrax bacilli growing in broth are destroyed by sanitas oil in the proportions—

1 : 200 and 1 : 150 after a quarter of an hour;

but that they are unaffected by solutions—

1 : 150 acting for one minute,

1 : 250 acting for one hour.

II. *Action on Anthrax Spores.*

1. 1 : 10.

A sterile test-tube was filled with 4½ c.c. resin-soap solution (20 grs. to 1 oz.). This was then well inoculated with anthrax spores from a potato culture, and sanitas oil was added in the above proportion. From this mixture a tube of broth and one of agar were inoculated after five minutes, a tube of broth and one of agar after a quarter of an hour, a tube of broth and one of gelatin after one hour, a tube of broth after five hours, and a tube of broth and one of gelatin after forty-eight hours. All the tubes showed a normal growth of anthrax twenty-four hours after they had been inoculated.

2. 1 : 5.

4 c.c. resin-soap solution were well inoculated with anthrax spores from a culture on potato, and the sanitas oil was added in the above proportion. Six tubes of broth inoculated from the mixture after five minutes, a quarter, half, one, five, and twenty-four hours respectively, all showed a normal growth of anthrax. In both these experiments with anthrax spores the oil of sanitas was present in too large a proportion to form a good emulsion, and a little of the pure oil gradually separated out and formed a thin layer on the top of the liquid in the test-tube.

Conclusions.—Anthrax spores are, therefore, unaffected by sanitas oil in the proportions 1 : 10 and 1 : 5 acting for twenty-four hours.

III. *Action on Staphylococcus pyogenes aureus.*

1. 1 : 75.

The emulsion of oil of sanitas was added in the above proportion to 5 c.c. broth containing a three days' growth of *Staphylococcus aureus*. From this mixture four tubes of broth and four of agar were inoculated, two at a time, after a quarter, half, one, and twenty-four hours respectively. All these tubes showed a normal growth of *Staphylococcus aureus* in two days' time.

2. 1 : 40.

Sanitas oil was added in the above proportion to 2 c.c. broth containing a growth of *Staphylococcus aureus* two days old. From this mixture a tube of broth and one of agar were inoculated after a quarter of an hour, a tube of agar after half an hour, and a tube of broth after one hour. In forty-eight hours all the tubes showed a good growth of *Staphylococcus aureus.*

3. 1 : 30.

Sanitas oil was added in the above proportion to 2 c.c. broth containing a twenty-four hours' growth of *Staphylococcus aureus.* A tube of agar was inoculated from the mixture after a quarter of an hour, a tube of broth after half an hour, a tube of agar after one hour, a tube of broth and one of agar after four hours, and a tube of broth after twenty-four hours. In all these tubes there occurred a normal growth of *Staphylococcus aureus.*

4. 1 : 20.

A sterile test-tube was filled with 4 c.c. resin-soap solution (20 grs. to 1 oz.). This was well inoculated with *Staphylococcus aureus* from a culture on the surface of agar, and oil of sanitas was added in the above proportion. From this mixture a tube of broth and one of gelatin were inoculated after a quarter of an hour, a tube of agar and one of gelatin after half an hour, a tube of gelatin after one hour, and a tube of agar after twenty-four hours. In all the tubes there occurred a good growth of *Staphylococcus aureus.*

Conclusions.—From these experiments it appears that *Staphylococcus pyogenes aureus* growing in broth is unaffected by oil of sanitas in the proportions—

1 : 30 and 1 : 20 acting for twenty-four hours.

Oleum Pini Sylvestris.

This substance, like oil of sanitas, was emulsified by means of resin soap so as to render it miscible with water and broth.

I. *Action on Sporeless Anthrax Bacilli.*

1. 1 : 200.

The emulsion of pine oil was added in the above proportion

to 10 c.c. broth containing a twenty-four hours' growth of anthrax bacilli.

From this mixture a tube of broth and one of agar were inoculated after a quarter of an hour, a tube of broth and one of agar after half an hour, and a tube of broth after one hour. All the tubes remained free from growth.

2. 1 : 150.

The emulsion was added in the above proportion to 10 c.c. broth containing a twenty-four hours' growth of anthrax bacilli.

Three tubes of broth and three of agar were inoculated from this mixture, two at a time, after one minute, a quarter, and half an hour respectively. All the tubes remained free from growth except the tube of broth inoculated after one minute, which showed a good growth of anthrax in twenty-four hours.

Conclusions.—These two experiments show that anthrax bacilli are destroyed by Oleum Pini Sylvestris in the proportions—

1 : 200 and 1 : 150 acting for a quarter of an hour.

II. *Action on Anthrax Spores.*

1 : 10.

A sterile test-tube was filled with 4½ c.c. resin-soap solution (20 grains to 1 oz.). This was well inoculated with anthrax spores from a potato culture, and the pine oil was added in the above proportion.

From this mixture a tube of broth and one of agar were inoculated after a quarter of an hour, a tube of broth and one of gelatin after one hour, a tube of broth after five hours, a tube of broth and one of gelatin after forty-eight hours. In all the tubes there occurred a normal growth of anthrax.

Conclusions.—An emulsion of Oleum Pini Sylvestris, therefore, acting for forty-eight hours, has no effect on anthrax spores.

Ipecacuanha.

I. *Action on Sporeless Anthrax Bacilli.*

1. Ten preliminary experiments were made, in which unmeasured quantities of ipecacuanha—weighing roughly from 5 to 10 grains—were added to tubes of peptone broth containing a twenty-four hours' growth of anthrax bacilli. From each of these tubes, after the ipecacuanha had acted for a quarter or half an hour, one or more fresh tubes of nutrient media were inoculated. Twenty tubes in all were so inoculated, and of these, six remained free from growth of any kind; the other fourteen all became impure, owing to the growth of the bacilli which had been introduced with the ipecacuanha. In none of the tubes were any anthrax bacilli ever found on microscopical examination.

2. 5 grains.

In four experiments 5 grains of ipecacuanha were weighed out and added to 5 c.c. broth containing a twenty-four hours' growth of anthrax bacilli free from spores.

Exp. A.—From the first tube containing this mixture six tubes of broth were inoculated, two at a time, after a quarter, half, and two hours respectively.

After having been twenty-four hours in the incubator, no growth was visible in any of the tubes. After forty-eight hours, however, a thin small bacillus was found to be growing in five out of the six tubes, and on the fourth day this bacillus had formed a thick pellicle covering the surface of the broth. In none of the tubes were any anthrax bacilli found at any time on microscopical examination.

Exp. B.—From the second tube containing the mixture two tubes of agar were inoculated after half an hour. In neither of the tubes was there any growth of anthrax, but in one of them a pellicle gradually formed, due to the presence of the same small bacillus as in Exp. A.

Exp. C.—From the third tube a mouse was inoculated after a quarter of an hour, and a tube of broth and one of agar were inoculated after half an hour. The mouse remained alive and well. Both the tubes became impure, and under

the microscope the same bacillus was seen as in the two preceding experiments, but no anthrax bacilli could be found. A mouse, which had been inoculated from the above tube before adding the ipecacuanha, was found dead and cold in forty-eight hours, and its blood was swarming with anthrax bacilli.

Exp. D.—From the fourth tube containing the mixture two tubes of gelatin were inoculated after a quarter and half hour respectively. The gelatin in these tubes was then melted by placing them for a little time in the incubator at 35° C., and the contents of each tube were poured out on to two glass plates placed in a damp chamber. After two days the plates were examined under the microscope, and numerous colonies of motile and non-motile bacilli were found, but neither then nor subsequently were any colonies of anthrax bacilli seen.

3. 3 grains.

In two experiments 3 grains of ipecacuanha were added to 5 c.c. broth containing a twenty-four hours' growth of anthrax bacilli.

Exp. A.—From a tube containing this mixture a tube of broth and one of gelatin were inoculated after a quarter of an hour, and a tube of broth and one of agar after one hour. In none of these tubes was there any growth of anthrax bacilli, but they all became impure after twenty-four hours, and both motile and non-motile bacilli were found on microscopic examination.

Exp. B.—From a second tube containing this mixture two tubes of gelatin were inoculated after the ipecacuanha had acted for half an hour. The gelatin in these tubes was then melted, and the contents were poured out on to two glass plates placed in a damp chamber. Each of these plates showed, in two or three days, several colonies of different bacilli and micrococci, but there were no spots of anthrax.

4. 2 grains.

Two grains of ipecacuanha were added to 5 c.c. broth containing a twenty-four hours' growth of anthrax bacilli. From this mixture a tube of agar and one of gelatin were inoculated after a quarter of an hour, and a tube of broth and one of gelatin after half an hour. Three of the tubes became impure, but none of them showed any growth of anthrax.

5. 1 grain.

One grain of ipecacuanha was added to 5 c.c. broth, containing a twenty-four hours' growth of anthrax bacilli free from spores. From this mixture two tubes of gelatin were inoculated after a quarter and half an hour respectively. The gelatin in these tubes was melted in the incubator, and the contents were then poured out on two glass plates placed in a damp chamber. In three days' time each plate showed numerous round colonies, which on microscopical examination were found to consist of anthrax bacilli.

6. ½ grain.

Half a grain of ipecacuanha was added to 5 c.c. broth containing a twenty-four hours' growth of anthrax bacilli. A tube of gelatin inoculated from this mixture after one minute, and two tubes of gelatin and one of agar inoculated after half an hour, all showed a normal growth of anthrax.

II. *Action on Anthrax Spores.*

5 grains.

A sterile test-tube was filled with 5 c.c. sterilised distilled water; this was well inoculated with anthrax spores taken from a culture on potato, and five grains of powdered ipecacuanha were then added. From this mixture a tube of agar was inoculated after a quarter of an hour, a tube of broth and one of gelatin after half an hour, a tube of agar after one hour, a tube of broth after three hours, and another tube of broth after twenty-four hours. All these tubes showed next day a normal growth of anthrax bacilli, but three of the tubes afterwards became impure.

Conclusions.—The numerous experiments above recorded show that ipecacuanha in small quantities has a marked power of destroying sporeless anthrax bacilli, but that it has no action on anthrax spores. The smallest quantity of the powder that would destroy all the bacilli contained in a 5 c.c. broth culture twenty-four hours old was found to be two grains. When smaller quantities, such as one or a half grain, were added, the bacilli were found to be still alive after half an hour.

According to the British Pharmacopœia, the active con-

stituents of ipecacuanha are an alkaloid emetine forming ½ to 1 per cent. of the root, and an organic acid called cephaëlic or ipecacuanhic acid. Of these two bodies emetine is usually considered to be the more important, and it therefore seemed probable that the action of ipecacuanha on anthrax bacilli might be due to the presence in it of this alkaloid.

Mr. Collier very kindly procured for me a pure specimen of emetine. This was dissolved in distilled water by the aid of a little dilute acetic acid, and with the solution so obtained the following experiments were made.

Emetine.

1. 1 : 200.

The solution of emetine was added in the above proportion to 5 c.c. broth containing a twenty-four hours' growth of anthrax bacilli free from spores. A tube of broth and one of agar were inoculated from the mixture after a quarter of an hour, a mouse was inoculated after half an hour, and a tube of broth and one of gelatin after three quarters of an hour.

The four tubes of nutrient media all showed a good growth of anthrax next day. The mouse was found dead on the third day, and its blood was swarming with anthrax bacilli.

2. 1 : 150.

Emetine was added in the above proportion to 10 c.c. broth containing a twenty-four hours' growth of anthrax bacilli. A tube of broth and one of gelatin inoculated from this mixture after a quarter of an hour showed a good growth of anthrax bacilli next day. A mouse inoculated after half an hour died in forty-eight hours, and its blood was found to contain numerous anthrax bacilli.

3. 1 : 100.

Emetine was added in the above proportion to 5 c.c. broth containing a twenty-four hours' growth of anthrax bacilli. A tube of broth and one of agar inoculated from this mixture after a quarter of an hour, and a tube of agar and one of gelatin inoculated after half an hour, all showed normal growth of anthrax next day.

Conclusions.—The result of these experiments is to show that emetine, even in the comparatively strong proportions of

1 : 100, has no action on sporeless anthrax bacilli. The power possessed by ipecacuanha of destroying these bacilli must, therefore, be due to some other constituent, and with regard to this point further experiments are needed.

B. Experiments to Determine the Strength of the Antiseptics necessary to Prevent the Growth of the Micro-organisms.

Corrosive Sublimate.

For each experiment a measured quantity of the stock solution (1 : 50) was diluted with sterilised distilled water in such a manner that $1\frac{1}{10}$ c.c. added to 10 c.c. peptone broth gave a mixture containing corrosive sublimate in the required proportion. The precautions necessary to prevent access of organisms from the air were the same as those taken in the first series of experiments.

I. *Action on the Growth of Anthrax Bacilli.*

1. 1 : 40,000.

Corrosive-sublimate solution was added to two test-tubes, each containing 10 c.c. peptone broth, so as to form a solution 1 : 40,000. The tubes were then well inoculated with anthrax from a potato culture, which consisted chiefly of spores, and were placed in the incubator. Both the tubes remained free from growth.

2. 1 : 50,000.

Sublimate solution was added to four tubes of broth in the proportion 1 : 50,000. Two of these tubes were inoculated with anthrax from a potato culture containing spores ; two of them from a broth culture of anthrax. All the tubes remained free from growth.

3. 1 : 70,000.

Sublimate solution was added to four tubes of broth in the proportion 1 : 70,000, and they were then inoculated with anthrax from potato cultures containing spores. All the tubes remained free from growth.

4. 1 : 75,000.

Sublimate solution was added to four tubes of broth in the proportion 1 : 75,000. Two of the tubes were inoculated with anthrax from a broth culture, and remained free from growth. The other two tubes were inoculated from a potato culture containing spores, and in three days each showed a rather scanty growth of anthrax bacilli.

5. 1 : 100,000.

Sublimate solution was added to four tubes of broth in the proportion 1 : 100,000. Two of the tubes were inoculated from a broth culture of anthrax, and remained free from growth. Two tubes were inoculated from a potato culture containing spores, and in twenty-four hours both of them showed a good growth of anthrax.

6. 1 : 200,000.

Sublimate solution was added to two tubes of broth in the proportion 1 : 200,000. They were then inoculated with anthrax from a broth culture, but neither of them afterwards showed any growth.

7. 1 : 300,000.

8. 1 : 500,000.

Sublimate solution was added to three tubes of broth in the proportion 1 : 300,000, and to three other tubes of broth in the proportion 1 : 500,000. These tubes were inoculated with anthrax from broth cultures, but none of them afterwards showed any growth.

9. 1 : 1,000,000.

On two occasions sublimate solution was added to two tubes of broth in the above proportion. The tubes were inoculated with anthrax from broth cultures, and in each case a good growth occurred in twenty-four hours.

Conclusions.—From these experiments it appears that corrosive sublimate solutions 1 : 70,000 prevent the growth in broth both of anthrax spores from a potato culture and of anthrax bacilli taken from a broth culture. A solution 1 : 75,000 completely prevents the growth of anthrax bacilli from a broth culture, but only retards the growth of anthrax spores. A solution 1 : 100,000 prevents growth in tubes of broth inoculated with bacilli from broth cultures, but has no effect on the growth of anthrax in tubes inoculated with spores

from a potato culture. Solutions 1 : 300,000 and 1 : 500,000 still prevent the growth of anthrax in tubes inoculated with the bacilli from broth cultures. Solutions 1 : 1,000,000 have no retarding effect on the growth of anthrax bacilli.

II. *Action on the Growth of Staphylococcus pyogenes aureus.*

1. In four preliminary experiments corrosive-sublimate solution was added to tubes containing 10 c.c. broth in the proportions 1 : 20,000, 1 : 25,000, 1 : 30,000, 1 : 40,000, two tubes being used for each experiment. These tubes were then well inoculated with *Staphylococcus aureus* from broth cultures, and were placed in the incubator. All the tubes remained free from growth.

2. 1 : 50,000.

Sublimate solution was added to four tubes of broth in the proportion 1 : 50,000. They were then inoculated with *Staphylococcus aureus* from a broth culture, but all of them remained free from growth.

3. 1 : 75,000.

Sublimate solution was added to two tubes of broth in the proportion 1 : 75,000. They were then inoculated with *Staphylococcus aureus* from a broth culture. In three days both of them showed a good growth of the organism.

4. 1 : 100,000.

Two tubes of broth, to which sublimate solution had been added in the proportion 1 : 100,000, were inoculated with *Staphylococcus aureus* from a broth culture. Both the tubes showed a good growth of the organism in two days.

Conclusions.—From these experiments it appears that corrosive sublimate in the proportion 1 : 50,000 completely prevents the growth of *Staphylococcus aureus* in 10 c.c. peptone broth, but in the proportion 1 : 75,000 it has no effect.

Carbolic Acid.

I. *Action on the Growth of Anthrax Bacilli.*

1. 1 : 500.

Carbolic acid solution was added in the above proportion

to four tubes, each containing 10 c.c. peptone broth. Two of these tubes were inoculated with anthrax from a potato culture containing spores; the other two tubes were inoculated from a recent broth culture. All the tubes remained free from growth.

2. 1 : 1000.

Carbolic acid solution was added in the above proportion to four tubes of broth. Two of these were inoculated from a potato culture containing spores, the other two tubes were inoculated from a broth culture. In twenty-four hours all the tubes showed a good growth of anthrax.

Conclusions.—Carbolic acid, therefore, in the proportion 1 : 500 completely prevents the growth of anthrax in peptone broth, whether the tubes be inoculated from a broth or a potato culture, whilst in the proportion 1 : 1000 it has no effect.

II. *Action on the Growth of Staphylococcus pyogenes aureus.*

1. 1 : 300.

Carbolic acid was added in the above proportion to three tubes of broth, and these were then inoculated with *Staphylococcus aureus* from broth cultures. All the tubes remained free from growth.

2. 1 : 500.

Carbolic acid was added to two tubes of broth in the above proportion. The tubes were then inoculated with *Staphylococcus aureus* from a broth culture, and in twenty-four hours both of them showed a good growth of the organism.

Conclusions.—Carbolic acid, therefore, in the proportion 1 : 300 prevents the growth in broth of *Staphylococcus pyogenes aureus*, but in the proportion 1 : 500 it has no effect.

Creolin.

I. *Action on the Growth of Anthrax Bacilli.*

1. 1 : 4000.

Creolin was added in the above proportion to two tubes,

each containing 10 c.c. broth, and they were then inoculated with anthrax bacilli from broth cultures. Both the tubes remained free from growth.

2. 1 : 5000.

Creolin was added in the above proportion to four tubes of broth, and they were then inoculated with anthrax from broth cultures. All the tubes remained free from growth.

3. 1 : 10,000.

Creolin was added in the above proportion to four tubes of broth, and they were then inoculated with anthrax from broth cultures. All the tubes showed a good growth of anthrax in twenty-four hours.

Conclusions.—From these experiments it appears that creolin in the proportion 1 : 5000 prevents the growth of anthrax bacilli in peptone broth, but that in the proportion 1 : 10,000 it has no effect.

II. *Action on the Growth of Staphylococcus pyogenes aureus.*

1. 1 : 2500.

Creolin was added in the above proportion to five tubes, each containing 10 c.c. peptone broth, and they were then inoculated with *Staphylococcus aureus* from broth cultures. All the tubes remained free from growth.

2. 1 : 4000.

Creolin was added in the above proportion to two tubes of broth, and they were then inoculated with *Staphylococcus aureus* from broth cultures. Both the tubes showed an active growth in twenty-four hours.

3. 1 : 5000.

Two tubes of broth containing creolin in the above proportion were inoculated with *Staphylococcus aureus* from a broth culture. In twenty-four hours both the tubes showed an active growth of the organism.

Conclusions.—Creolin, therefore, in the proportion 1 : 2500 prevents the growth of *Staphylococcus pyogenes aureus*, but has no effect in the proportion 1 : 4000.

Coal-tar Emulsion (Liquor Picis Carbonis).

I. *Action on the Growth of Anthrax Bacilli.*

1. 1 : 5000.

The emulsion was added in the above proportion to two tubes, each containing 10 c.c. peptone broth; the tubes were then inoculated with anthrax from a recent broth culture, but both remained free from growth.

2. 1 : 10,000.

Two tubes of broth, containing the emulsion in the above proportion, were inoculated with anthrax from a broth culture, and in twenty-four hours showed an active growth.

Conclusions.—Coal-tar emulsion, therefore, in the proportion 1 : 5000 prevents the growth in broth of anthrax bacilli, but in the proportion 1 : 10,000 it has no effect.

II. *Action on the Growth of Staphylococcus pyogenes aureus.*

1. 1 : 2000.

Coal-tar emulsion was added in the above proportion to four tubes, each containing 10 c.c. peptone broth; these were then inoculated with *Staphylococcus aureus* from broth cultures. All the tubes remained free from growth.

2. 1 : 3000.

Two tubes of broth containing the emulsion in the above proportion were inoculated with *Staphylococcus aureus* from broth cultures. After twenty-four hours both tubes showed a good growth of the organism.

Conclusions.—Coal-tar emulsion, therefore, in the proportion 1 : 2000 prevents the growth of *Staphylococcus aureus*, but in the proportion 1 : 3000 it has no effect.

Oil of Sanitas.

I. *Action on the Growth of Anthrax Bacilli.*

1. 1 : 4000.

Oil of sanitas was added in the above proportion to four

tubes, each containing 10 c.c. broth; these were then inoculated with anthrax from a broth culture. All remained free from growth.

2. 1 : 6000.

Four tubes of broth containing oil of sanitas in the above proportion were inoculated with anthrax from a broth culture. After twenty-four hours all the tubes showed a good growth of the organism.

Conclusions.—The growth of anthrax bacilli in broth is, therefore, prevented by oil of sanitas when present in the proportion 1 : 4000, but in the proportion 1 : 6000 it has no effect.

II. *Action on the Growth of Staphylococcus pyogenes aureus.*

1. 1 : 1000.

Two tubes of broth containing oil of sanitas in the above proportion were inoculated with *Staphylococcus aureus* from a broth culture. Both the tubes remained free from growth.

2. 1 : 2000.

Two tubes of broth containing oil of sanitas in the above proportion were inoculated with *Staphylococcus aureus* from a broth culture. In twenty-four hours both the tubes showed a good growth of the organism.

3. 1 : 2500.

Two tubes of broth containing oil of sanitas in the above proportion were inoculated with *Staphylococcus aureus* from a broth culture, and in twenty-four hours both showed a good growth of the organism.

Conclusions.—Oil of sanitas, therefore, in the proportion 1 : 1000 prevents the growth of *Staphylococcus aureus* in peptone broth, but in the proportion 1 : 2000 it exerts no effect.

Oleum Pini Sylvestris.

Action on the Growth of Anthrax Bacilli.

1. 1 : 4000.

Oleum Pini Sylvestris was added in the above proportion

to two tubes, each containing 10 c.c. broth; they were then inoculated with anthrax from a broth culture, but both tubes remained free from growth.

2. 1 : 6000.

Oleum Pini Sylvestris was added in the above proportion to four tubes of broth, and they were then inoculated with anthrax bacilli from broth cultures. In twenty-four hours all the tubes showed an active growth.

Conclusions.—It appears, therefore, that *Oleum Pini Sylvestris* in the proportion 1 : 4000 prevents the growth of anthrax bacilli in peptone broth, but that in the proportion 1 : 6000 it has no effect.

	Sporeless anthrax bacilli.			Anthrax spores.			Staphylococcus pyogenes aureus.		
	Strength of solution.	Time in which the bacilli were killed.	Time after which bacilli were found to be still alive.	Strength of solution.	Time in which the spores were killed.	Time after which spores were found to be still alive.	Strength of solution.	Time in which the Staphylococci were killed.	Time after which the Staphylococci were found to be still alive.
Corrosive sublimate	1 : 30,000	—	1 hour	1 : 5000	—	1 hour	1 : 3000	—	24 hours
	1 : 25,000	½ hour	¼ hour	1 : 3000	1 hour	½ hour	1 : 1000	—	24 hours
	1 : 20,000	½ hour	¼ hour	1 : 1500	1 hour	½ hour	1 : 600	—	1 hour
	1 : 15,000	1 minute	—	1 : 1000	¼ hour	3 minutes	1 : 500	—	1 hour
							1 : 250	—	½ hour
							1 : 200	¼ hour	—
							1 : 150	¼ hour	—
Carbolic acid	1 : 300	—	1 hour	1 : 100	—	3 hours	1 : 50	½ hour	—
	1 : 175	½ hour	¼ hour	1 : 20	24 hours	4 hours	1 : 40	½ hour	—
	1 : 150	¼ hour	1 minute						
	1 : 100	5 minutes	—						
Creolin	1 : 500	—	4 hours	1 : 30	—	6 days	1 : 100	24 hours	1 hour
	1 : 400	—	1 hour	1 : 10	—	2 days	1 : 75	¼ hour	—
	1 : 300	1 minute	—				1 : 50	¼ hour	—
	1 : 200	1 minute	—						
Coal-tar emulsion (Liquor Picis Carbonis)	1 : 500	—	4 hours	1 : 30	—	6 days	1 : 75	—	2 days
	1 : 400	1 hour	½ hour	1 : 10	—	2 days	1 : 40	—	24 hours
	1 : 300	1 minute	—						
	1 : 200	1 minute	—						
Oil of sanitas	1 : 250	—	1 hour	1 : 10	—	24 hours	1 : 30	—	24 hours
	1 : 200	¼ hour	1 minute	1 : 5	—	24 hours	1 : 20	—	24 hours
	1 : 150	¼ hour	1 minute						
Oleum Pini Sylvestris	1 : 200	¼ hour	1 minute	1 : 10	—	48 hours			
	1 : 150	¼ hour	1 minute						

B. *Table showing the Retaining Power of the Antiseptics on the Growth of the Micro-organisms.*

	Anthrax bacilli.		Staphylococcus pyogenes aureus.	
	Prevents growth.	Does not prevent growth.	Prevents growth.	Does not prevent growth.
Corrosive sublimate	1 : 500,000 (bacilli) 1 : 70,000 (spores)	1 : 1,000,000 (bacilli) 1 : 75,000 (spores)	1 : 50,000	1 : 75,000
Carbolic acid	1 : 500	1 : 1000	1 : 300	1 : 500
Creolin	1 : 5000	1 : 10,000	1 : 2500	1 : 4000
Coal-tar emulsion (Liquor Picis Carbonis)	1 : 5000	1 : 10,000	1 : 2000	1 : 3000
Oil of sanitas	1 : 4000	1 : 6000	1 : 1000	1 : 2000
Oleum Pini Sylvestris	1 : 4000	1 : 6000	—	—

From the results of my experiments set forth in the preceding tables, there are, I think, some important and interesting conclusions to be drawn as to the relative value of the different antiseptic substances tested, especially in relation to their practical use in surgery and medicine.

Corrosive sublimate still stands at the head of the list in regard to its actual power both as a disinfectant and as an antiseptic.

Easily killed organisms, as represented by anthrax bacilli free from spores, are destroyed by such dilute solutions as 1 : 15,000 acting for one minute, and 1 : 20,000 acting for half an hour.

The growth of the bacilli in peptone broth is entirely prevented when sublimate is added to it in the proportion 1 : 500,000.

Anthrax spores, which are very resistant to all forms of disinfection, are killed by a solution 1 : 1000 acting for a quarter of an hour.

The growth of the spores in broth is completely prevented when sublimate is present in the proportion 1 : 70,000.

These facts are, however, of only indirect importance in regard to the practical value of corrosive sublimate as a disinfectant and antiseptic. The bacilli and spores of anthrax are only exceptionally met with in surgery and medicine, and

we can merely infer from the above results that if sublimate has such a great power of destroying and preventing the growth of a resistant organism like anthrax spores, it will have an equally powerful action on the organisms ordinarily present in wounds, secretions, on instruments, hands, &c.

The experiments with *Staphylococcus pyogenes aureus*, on the other hand, have a direct practical bearing.

This organism has a close connection with most forms of suppuration, pyæmia, and septicæmia; it is, therefore, most important, from a practical point of view, that an antiseptic substance should have great power, not only of preventing the growth of *Staphylococcus aureus*, but also of actually destroying it.

The results of experiments with this organism have probably a further importance, for Tarnier and Vignal, in a recent research on antiseptics, quoted fully further on, have found that the *Streptococcus pyogenes* is destroyed, and its growth is prevented by exactly the same strengths of different antiseptic solutions as are effective against *Staphylococcus aureus*. This Streptococcus is a still more dangerous organism than the Staphylococcus, playing as it does an active part in the causation of ulcerative endocarditis, and the worst forms of puerperal fever and septicæmia. If, therefore, the experiments with *Staphylococcus aureus* apply also to this Streptococcus, they will have a double value.

When the power possessed by corrosive sublimate of killing *Staphylococcus aureus* was tested, very unexpected results were obtained.

This organism, growing in peptone broth, was only completely destroyed by a corrosive-sublimate solution 1 : 200 acting for a quarter of an hour.

Weaker solutions, such as 1 : 600 and 1 : 500, impaired to some extent the vitality of the Staphylococci, but, even after they had acted for one hour, a fairly active growth was obtained when the organism was removed into a suitable culture medium.

With a solution 1 : 1000 no effect was observed on the vitality of the Staphylococci, even when exposed to it for twenty-four hours.

Now, although corrosive sublimate is sometimes used for

purifying a foul wound in as strong a solution as 1 : 500, yet 1 : 1000 is the strongest solution that it is safe to employ in any quantity, or for more than momentary application. A solution of this strength even is regarded by many surgeons as being dangerously strong for most purposes.

Hence, according to my experiments, corrosive-sublimate lotion of the ordinary strengths, 1 : 1000 to 1 : 5000, cannot be regarded as a reliable agent, where it is desired to destroy the organisms of suppuration or of blood-poisoning. For these purposes some of the other substances mentioned below are more suitable.

Although, however, corrosive sublimate is unable to destroy *Staphylococcus aureus* except when used in poisonous quantities, it possesses a very considerable power of preventing its growth in nutrient media. For the growth of this organism in broth is completely prevented when sublimate is added in the proportion 1 : 50,000.

Where, therefore, it is merely desired to prevent the development of the organisms of suppuration, as, for example, in dressing a clean wound, corrosive sublimate in very dilute solutions is probably the best substance to employ.

In reference to the feeble power shown by sublimate of destroying *Staphylococcus aureus*, it is interesting to note its still weaker action on *yellow sarcina*, an organism not in any way pathogenic or hurtful, but which morphologically, at any rate, belongs to the micrococci.

Yellow sarcina growing in peptone broth is completely destroyed by corrosive sublimate 1 : 100, acting for half an hour. Its vitality is merely impaired by solutions 1 : 300 and 1 : 500 acting for one hour.

Carbolic acid was the next substance tested, and on the whole came out the best.

Anthrax bacilli, free from spores, are destroyed by carbolic acid 1 : 175 acting for half an hour, and by a 1 per cent. solution acting for one minute. The spores of anthrax show a considerable power of resistance to its action, as they are only killed by a 5 per cent. solution after being exposed to it for twenty-four hours.

The growth in broth of the spores and bacilli of anthrax is

prevented by carbolic acid, when present in the proportion 1 : 500. The great value of carbolic acid, however, lies in its action on *Staphylococcus pyogenes aureus*. This organism growing in broth is killed by carbolic acid in the proportion 1 : 50 acting for a quarter of an hour. A 2 per cent. solution may, therefore, be regarded as a thoroughly reliable disinfectant, and in this strength it is sufficiently non-poisonous and non-irritating to be used for nearly all purposes. The growth of *Staphylococcus pyogenes aureus* in broth is prevented when carbolic acid is present in the proportion 1 : 300. Where, therefore, an antiseptic only is required, as opposed to a disinfectant, it may be regarded as inferior in value to corrosive sublimate; it is, moreover, not so suited as the latter for preventing the development of organisms during any lengthened period of time, both on account of its volatility and of its slightly irritant action on the skin even in dilute solutions.

Creolin in many respects appears to be a more powerful disinfectant and antiseptic than carbolic acid. Sporeless anthrax bacilli are destroyed by a solution of creolin 1 : 300 acting for one minute. The growth of the bacilli in broth is prevented when creolin is added in the proportion 1 : 5000. Anthrax spores are not completely killed by a 10 per cent. solution acting for two days. The virulence of the spores would, however, seem to be impaired at a much earlier period by this 10 per cent. solution, as two guinea-pigs, inoculated in different experiments with anthrax spores which had been exposed to its action for only half an hour, remained alive and unaffected.

Staphylococcus aureus is killed by a 1 per cent. solution of creolin acting for twenty-four hours, and by a solution 1 : 75 acting for a quarter of an hour, whilst the growth of the organism in broth is prevented when creolin is added in the proportion 1 : 2500. This substance, therefore, may be considered thoroughly efficient both as a disinfectant and antiseptic. Its use, however, must be limited to some extent by the fact that it forms an opaque emulsion with water, so that, when used as an application to wounds, &c., it tends to obscure any changes going on in the part; it is, moreover,

very liable, when kept for a short time, to undergo decomposition, with formation of a black tarry precipitate.

Coal-tar emulsion, corresponding to Liquor Picis Carbonis, was tested in the hope of finding a preparation of known composition which might take the place of the patented liquid, creolin. Anthrax bacilli free from spores are killed by a solution of the emulsion in the proportion 1 : 300 acting for one minute, and the development of the bacilli in broth is prevented when the emulsion is added in the proportion 1 : 5000. Anthrax spores are not completely destroyed by a 10 per cent. solution acting for two days. The emulsion is, however, much inferior to creolin in its action on *Staphylococcus pyogenes aureus*. This organism is not killed by a solution 1 : 40 acting for twenty-four hours, although the growth of the Staphylococcus in broth is prevented when the emulsion is added in the proportion 1 : 2000. Like creolin, this substance forms an opaque emulsion with water, but it does not decompose so readily.

Mr. Collier tells me that an emulsion could easily be made containing a larger proportion of prepared coal-tar than the above; and it is, I think, quite likely that in this way a liquid might be obtained which should be as good a disinfectant and antiseptic as creolin.

Oil of sanitas and *Oleum Pini Sylvestris* are probably of about equal power in their action on micro-organisms. Both of these substances destroy anthrax bacilli in solutions of strength 1 : 200 acting for a quarter of an hour, and they prevent the growth of the bacilli in broth when added in the proportion 1 : 4000. Both, also, even in the proportion 1 : 10, when allowed to act on anthrax spores for twenty-four hours, are without effect. The vitality of *Staphylococcus aureus* is not affected by oil of sanitas in the proportion 1 : 20 acting for twenty-four hours, but the development of the organism in broth is prevented when the oil is added in the proportion 1 : 1000. The action of Oleum Pini Sylvestris on *Staphylococcus aureus* was not tried.

These two substances, therefore, are probably of little use for disinfecting purposes, but they may be of use as antiseptics.

They are pleasant smelling and are practically colourless, but they are very insoluble in water.

Having gone over the results of my own experiments, I will conclude this paper by a brief epitome of the results obtained by other observers in similar experiments with one or more of the same antiseptic substances.

An outline of the method employed in each case must also be given, as I hope to show that the different results obtained are largely due to differences in the method of experiment.

The first experiments as to the action of antiseptics on pure cultures of micro-organisms were those of *Koch*, published in the 'Mittheilungen aus dem Kaiserlichen Gesundheitsamte,' vol. i, 1881.

To determine the killing action of various substances on anthrax spores, Koch immersed silk threads, charged with the spores, in 20 c.c. of the antiseptic solutions. After certain intervals of time one or more of the threads was removed, and after being washed in water or alcohol, was planted in gelatin or other nutrient material, and the subsequent growth, if any, was noted.

In the case of sporeless anthrax bacilli silk threads were soaked in the spleen of a mouse which had died of anthrax; they were dried, and were then immersed in the antiseptic solutions in the same way as with the spores.

The following results were obtained with corrosive sublimate and carbolic acid.

Anthrax spores were killed by corrosive-sublimate solutions 1 : 1000 and 1 : 5000 when simply sprinkled with the liquid. They were killed in five minutes by a solution 1 : 10,000, and in ten minutes by a solution of 1 : 20,000. A solution 1 : 50,000 acting for one hour had no effect.

In these experiments the spores were thought to be killed when no growth occurred around the silk threads transplanted into gelatin.

Subsequently Koch found that this was not altogether to be relied on as a proof of their death, for in some other experiments, a mouse inoculated with anthrax spores, which had been acted on by sublimate 1 : 20,000 for ten minutes, died in four days.

Another mouse inoculated with spores acted on by sublimate 1 : 10,000 for ten minutes died in five days. A third mouse inoculated with spores acted on by sublimate 1 : 10,000 for one hour lived.

Hence he concludes that to destroy anthrax spores with certainty, corrosive sublimate should be used in the proportion 1 : 5000.

With carbolic acid the following results were obtained:— Anthrax spores were destroyed by a 3 per cent. solution of carbolic acid in seven days, by a 4 per cent. solution in three days, by a 5 per cent. solution in two days.

The vitality of the spores was, however, much impaired by the 5 per cent. solution in twenty-four hours.

Sporeless anthrax bacilli from the spleen of a mouse were killed by a 1 per cent. solution of carbolic acid in two minutes.

The action of carbolic acid on sporelesss anthrax bacilli was also tested by Koch in another way.

The blood of an animal dead of anthrax was mixed with an equal quantity of 1 per cent. carbolic acid, thus giving a solution 1 : 200. After a short time it was found that anthrax was no longer produced in other animals when they were inoculated from the mixture. A similar experiment was made in which the blood was mixed with an equal quantity of ½ per cent. carbolic acid, thus giving a solution 1 : 400. All the animals inoculated from this mixture died rapidly of anthrax.

As to the restraining action on the growth of anthrax, Koch found that corrosive sublimate 1 : 330,000 completely prevents the growth of anthrax in peptone broth, whilst in the proportion 1 : 600,000 it still exerts a distinctly retarding effect.

Carbolic acid 1 : 850 prevented the growth of anthrax in broth, and 1 : 1250 exerted a retarding effect.

As showing the greatest divergence from Koch's results I will next quote some experiments made by *Klein* on the action of corrosive sublimate, and published by him in the Local Government Board 'Reports,' 1885 to 1886.

To test the action of sublimate on anthrax spores, Klein

mixed a trace of spore material in a watch-glass with a large and in each case equal excess of sublimate solution of known strength; after a certain time a drop was removed from the mixture, and with it a tube of culture medium or a guinea-pig was inoculated.

By this method he found that anthrax spores grown for four weeks on agar were not completely killed by sublimate solution 1 : 1000 acting for one hour. Spores grown two months in broth were but little affected by sublimate solution 1 : 1000 acting for four days. Spores grown six months in broth were only slightly affected by sublimate solution 1 : 480 acting for five and fifteen minutes. The same spores were markedly weakened by solution 1 : 120 and 1 : 240 acting for five minutes, and they were completely killed by the same solutions acting for fifteen minutes.

Another method was employed to test the action of sublimate on sporeless anthrax bacilli and other organisms. A droplet of a broth culture of anthrax bacilli was allowed to ascend by capillarity into a freshly made glass pipette; into the same pipette the sublimate solution was also drawn up in such amount that the mixture in the pipette contained the original droplet diluted about 100 times by the antiseptic solution. After a given time tubes of nutrient media were inoculated from the mixture, and these were watched for any growth of anthrax.

In this way it was found that sporeless anthrax bacilli were unaffected by sublimate solution 1 : 25,000 acting for thirty-five and sixty minutes, but they were completely destroyed by sublimate solution 1 : 20,000 acting for thirty minutes.

As to the restraining action of sublimate, Klein found that the growth of the spores and bacilli of anthrax in gelatin was prevented when sublimate was added in the proportion 1 : 40,000, but that it was not hindered at all by sublimate in the proportion 1 : 50,000.

In 'Langenbeck's Archiv für klinische Chirurgie,' vol. xxxii, 1885, *Gärtner* and *Plägge* publish some experiments as to the disinfecting action of carbolic acid on various bacteria.

The organism to be tested was planted in a small flask con-

taining about 10 c.c. broth. It was allowed to grow for from twenty-four to seventy-two hours, and then 1 c.c. of the culture was removed and added to 49 c.c. of a 1, 2, or 3 per cent. solution of carbolic acid, whilst at the same time for comparison 1 c.c. of the culture was mixed with 49 c.c. of a 1 : 1000 solution of corrosive sublimate.

At intervals of time ranging from eight seconds to five minutes a drop was removed from these mixtures, and with it a tube of liquefied gelatin was inoculated. The gelatin was then poured out on to a glass plate placed in a damp chamber. This was watched, and the number of colonies that subsequently developed, if any, was counted.

The following results were obtained :—Sporeless anthrax bacilli are killed by 1 per cent. carbolic acid in fifteen seconds; *Staphylococcus pyogenes aureus* is unaffected by 1 per cent. carbolic acid acting for five minutes, but it is killed by 2 per cent. carbolic acid in eight seconds; *Streptococcus pyogenes* is killed by 1 per cent. carbolic acid in five minutes; *Staphylococcus pyogenes aureus* and *Streptococcus pyogenes* are both killed by 1 : 1000 corrosive sublimate in eight seconds.

In the 'Centralblatt für Bacteriologie und Parasitenkunde,' vol. ii, 1887, *von Esmarck* has published a series of experiments designed to test the disinfectant value of creolin as compared with carbolic acid. For this purpose flasks containing broth were inoculated from pure cultures of the micro-organisms, and were placed for twenty-four hours in the incubator. At the end of this time, a good growth of the organism having occurred, 10 c.c. of the culture were removed from the flask and were placed in two test-tubes; equal quantities of solutions of creolin and carbolic acid were then added, the contents of the tubes were well shaken, and after certain intervals of time, tubes containing liquefied gelatin were inoculated from the mixtures. These tubes were then made into roll cultures, *i. e.* the tube is held nearly horizontal, and is turned round and round until the gelatin in cooling solidifies in a uniform layer all over the inner surface. The number of colonies, if any, that afterwards developed in these tubes could thus be counted.

By this method it was found that a 1 per cent. solution of

creolin acting for ten minutes had no effect on *Staphylococcus pyogenes aureus;* after the same solution had acted for one hour the vitality of the organisms was somewhat impaired; after twenty-four hours the destructive effect was more marked, and after four days the Staphylococci were completely destroyed. On the other hand, a 1 per cent. solution of carbolic acid acting for four days had no effect at all on the Staphylococci.

The action on anthrax spores was tested by means of silk threads charged with the organisms, exactly as in Koch's method.

It was found that a 5 per cent. solution of creolin had little or no effect on anthrax spores after acting for six days, and even after twenty days they were not completely destroyed. A 5 per cent. solution of carbolic acid had no marked effect on anthrax spores after acting for four days, but they were completely destroyed after twenty days.

James Eisenberg ('Wiener medizinische Wochenschrift,' Nos. 17, 18, 19, 1888), following up the experiments of von Esmarck, tested the action of creolin on several different micro-organisms.

Five c.c. of a broth culture of the organism were mixed with an equal amount of a solution of creolin of known strength. From this mixture after certain intervals of time tubes of gelatin were inoculated. These tubes were then made into roll cultures, as in von Esmarck's experiments, and the number of colonies that afterwards occurred was noted.

In this way it was found that sporeless anthrax bacilli were killed by creolin 1 : 500 in two minutes; *Staphylococcus pyogenes aureus* had its vitality much impaired by creolin 1 : 100 after fifteen minutes; it was killed by creolin 2 : 100 in fifteen minutes, and by creolin 3 : 100 in one minute.

Experiments were also made with anthrax spores, by exposing silk threads charged with them to the action of creolin solutions for a given time. They were then taken out, washed in sterilised water, and planted in gelatin. It was thus found that anthrax spores were killed by creolin—6 per cent. in twenty-four hours; 3 per cent. in forty-eight hours, and 2 per cent. in ninety-six hours.

The spores were, on the other hand, unaffected by carbolic acid 8 per cent. acting for ninety-six hours.

With these results may be compared those obtained by *Dr. Washbourn*, and published by him in the 'Guy's Hospital Reports,' 1889.

The experiments were carried out by Koch's method; silk threads charged with anthrax spores being immersed in solutions of creolin for a given time, and then, after washing, planted in tubes containing beef broth.

Nine experiments were made, in each of which the anthrax spores were acted on by creolin 5 per cent. for five minutes. In every case the spores were entirely destroyed.

Nine comparative experiments were at the same time made, in which the anthrax spores were subjected for five minutes to the action of a 5 per cent. solution of carbolic acid.

In one experiment the spores were killed, but in all the others they were unaffected.

Tarnier and *Vignal*, in the 'Archives de Médicine Expérimentale et d'Anatomie Pathologique,' July, 1890, publish an interesting series of experiments on the action of some antiseptics on *Staphylococcus pyogenes aureus* and on *Streptococcus pyogenes*.

These two organisms were tested in parallel experiments throughout, and both gave the same results. To determine the power possessed by the antiseptics of killing the Staphylococci, they, in one series of experiments, inoculated flasks of broth with a drop of a culture of the organism forty-eight hours old.

These flasks were placed in the incubator for twenty-four hours, and the antiseptic was then added. Tubes of culture media were then from time to time inoculated from the mixture.

In this way they found that the Staphylococci were killed by

Corrosive sublimate	. 1 : 20,000	(0·5 grm. to 1 litre),
Carbolic acid	. 3 : 1000	(3 grms. „),
Creolin . . .	. 1 : 250	(4 „ „).

The time taken by these solutions to destroy the organisms is not stated.

In another series of experiments they exposed threads

charged with Staphylococci to the action of the antiseptic solutions, and found that the organisms were destroyed by—

Corrosive sublimate	.	1 : 1000 in 2 minutes.
„	.	1 : 4000 in 5 to 8 minutes.
Carbolic acid	.	1 : 100 in 15 to 17 minutes.
„	.	1 : 50 in 18 minutes.
Creolin	.	1 : 100 in 40 minutes.

But creolin 1 : 200 had no effect after acting for sixty minutes. To test the power possessed by the antiseptics of restraining the growth of *Staphylococcus aureus,* they inoculated, in each experiment, fifty grms. of peptone broth with two drops of a culture of the organism, and then added the antiseptic.

The development of *Staphylococcus aureus* was completely arrested by—

Corrosive sublimate	.	3 : 200,000 (·015 grm. to 1 litre),
Carbolic acid	.	1 : 500 (2 grms. „),
Creolin	.	3 : 1000 (3 „ „).

On looking through the results of the various experiments above quoted, it will be noticed that, whilst many of them agree fairly well together, others are more or less contradictory. The differences in the results are most marked in regard to the destroying action of corrosive sublimate, carbolic acid, and creolin on anthrax spores and *Staphylococcus pyogenes aureus.* Thus, to take only one example, Koch finds that anthrax spores are killed almost instantaneously by corrosive sublimate in the proportion 1 : 5000. According to my own experiments a solution of this strength has no effect on anthrax spores, but they are killed by a solution of sublimate 1 : 1000 acting for a quarter of an hour. Klein, however, states that, in some cases at least, anthrax spores are only completely destroyed by a sublimate solution 1 : 120 acting for fifteen minutes.

These differences are too frequent to be explained to any large extent by mere errors of observation or want of care in conducting the experiments. The explanation is, I think, to be found in part by considering the different methods employed in the various experiments.

To compare properly the results of any two observers in experiments with the same antiseptic substance acting on the same micro-organism, we must know—

First, the strength of the antiseptic solution employed.

Secondly, the time during which it acted.

Thirdly, the number of individual organisms which were present in a given quantity of the solution.

The probable importance of this third factor in relation to the results of the experiments may be shown by taking an imaginary case. Suppose two experiments are made to test the action of a corrosive-sublimate solution 1 : 1000 on anthrax spores. In the first experiment, say 100 spores are subjected for a quarter of an hour to the action of 5 c.c. sublimate solution 1 : 1000. In this case there will obviously be one twenty-thousandth part of a grain of pure mercuric chloride ($HgCl_2$) free to act on each individual spore.

In the second experiment, say one million spores are acted on for a quarter of an hour by 5 c.c. of the 1 : 1000 solution. Here there will only be one two-hundred-millionth part of a grain of mercuric chloride free to act on each spore, supposing all the spores are equally affected. That these two experiments with corrosive sublimate 1 : 1000 acting for a quarter of an hour should give entirely different results would hardly be surprising.

Now in all the experiments quoted in this paper—including my own—it will be seen that whilst the strength of the antiseptic, and, in most cases, the time during which it acted, are accurately recorded, no estimate is made in any experiment of the number of individual organisms that was acted on by a given quantity of the antiseptic solution. That the number must have varied enormously in the experiments of different observers is, however, clear from the account of the methods employed in each case.

In my own experiments with sporeless anthrax bacilli the organisms were in every case grown for twenty-four hours in 10 c.c. broth, and the quantity of the antiseptic fluid added to make a solution of the required strength varied between 1 and 2 c.c. The actual number of organisms present in the tubes was never estimated, but as each tube was placed under exactly the same conditions, it is probable that it varied but slightly, and the experiments are therefore quite comparable *inter se.*

In the experiments with anthrax spores, the number of

spores present in each tube was probably not quite so uniform, but it was always so great that a single drop of the fluid, examined under a one-sixth inch objective, showed crowds of spores in each field of the microscope, and so, with a total of some millions in every case, a considerable variation would make little practical difference.

The same remarks apply to the experiments with *Staphylococcus pyogenes aureus*. The total number of organisms present in the fluid was always very large, so that the presence of several thousands less or more in any individual tube would make but little difference.

Hence, my results show what strength of an antiseptic solution is required in order that a limited quantity of it may destroy a particular organism when present in very large numbers.

The method most generally employed by other observers has been that introduced by Koch. Silk threads are soaked in a liquid containing the organisms to be tested; a certain number of the organisms cling to the threads, and the latter are then taken out, dried, and kept in sterile vessels until required for use. Dr. Washbourn, who has some experience of this process in the case of anthrax spores, tells me that at first he had great difficulty in fixing the spores on the threads, and that even then, unless great care be taken, all the spores may be washed off by the antiseptic fluid. The quantity of antiseptic solution in which these threads are immersed is usually 20 c.c., so that here evidently the organisms are few and scattered, whilst the antiseptic solution is in relatively large excess.

Experiments carried out by Koch's method, therefore, show what strength of an antiseptic solution is required in order to destroy a particular organism when present in small numbers.

From these considerations we should expect to find that corrosive sublimate, carbolic acid, creolin, &c., when made to act on anthrax spores or *Staphylococcus pyogenes aureus* according to Koch's method, would be able to kill these organisms in much weaker solutions than when tested according to my own method. On looking back over the preceding pages, this will be found actually to be the case with but few exceptions.

The sets of experiments are, therefore, in no wise contradictory one to the other, but the results differ merely because they prove two different series of facts.

In conclusion, I must express my indebtedness to Mr. Collier for providing me with specimens of the various antiseptics that I required, and to Dr. Washbourn, who has helped me most materially, not only in the carrying out of my experiments, but also in the more arduous task of writing this paper about them.

CONGENITAL WRY-NECK

(CAPUT OBSTIPUM CONGENITALE; TORTICOLLIS CONGENITALIS);

WITH REMARKS ON FACIAL HEMIATROPHY.

By C. H. GOLDING-BIRD.

All the surgical text-books treat more or less fully of congenital wry-neck, but a series of six consecutive cases has shown me that more yet remains to be said upon the subject; or, at least, that points which are usually but mentioned in a casual fashion deserve a more careful study. Such a point, for instance, is the facial hemiatrophy which accompanies this malady, and which, for the most part, seems to be regarded as an accidental condition; it is, in reality, not only an essential part of congenital wry-neck, but in point of pathological evolution, I think, even its antecedent.

Then, again, those writers who have mentioned the side on which the disease appears, state that it usually affects the right sterno-mastoid; but I think that this is but part of the whole truth. In all the cases I have seen or read of the wry-neck has always been upon the right side, and every picture of congenital wry-neck that is in our text-books represents the face as looking over the left shoulder. I think, therefore, the statement that congenital wry-neck never affects the left side to be the correct one to make.

Use has been made of the fact of the frequency of the

malady being on the right side to explain its origin, but this will be discussed further on.

Congenital wry-neck is not a common condition, though in the course of years the surgeon is sure to meet with several examples, and its symptoms would not need description here save that it requires to be differentiated from another condition seen in infants, and at times confounded with wry-neck.

Cases of acquired wry-neck due to peripheral causes, to spinal disease, or any other cause operating some time after birth, are not to be classed as congenital; nor cases of clonic spasm of the sterno-mastoid.

Congenital wry-neck, from the history parents give of it, would not appear to be in reality a congenital condition. In all but one of the six cases that came under my own care I was informed that the deformity had only been recently noticed, and that even in the case of the oldest child, who was nearly ten years of age when he was presented for treatment. Two circumstances prove that the parents are deceived, strange though it may be that such a marked deformity could have passed so long unnoticed. One of these is an appeal to the family photograph album, and in three of my cases I was able to point out that the deformity existed years before the parents noticed it, it being quite evident in the earliest photographs of the children. The other proof is found in the fact that accompanying the wry-neck there is always more or less marked facial hemiatrophy of the right side, and that not merely of the muscles, as though from some muscular paralysis, but also of the other facial tissues. A measurement taken from the eyebrow to the lower jaw on the right side may be even a quarter of an inch less than that on the opposite side; the right cleft of the mouth is smaller than the left, and the patient talks "obliquely;" and the right external ear may be as much as a quarter of an inch smaller, in vertical measurement, than the left. Such a diminution of size could not occur within the short period of time allotted to the onset of the disease by the friends, but clearly antedates the history by many years—at least in the case of children of seven or eight.

The frequency with which the want of symmetry of the

face is overlooked is quite astonishing; and even when pointed out to parents they do not always observe it until they are shown the reflection of the face in a looking-glass. At once the slightest degree of want of symmetry of the features is by this means made apparent. A photograph similarly makes evident the facial deformity, and is credited with exaggerating it.

That facial hemiatrophy of the right side is much more frequent than would be supposed, I feel confident, and after a little practice with the mirror it becomes easy to recognise only a slight degree of asymmetry. I was much struck with this only lately when examining a number of asylum children. They had all passed the inspection of the matron, and been examined by two of the medical officers without a remark, but I at once detected in a girl of about ten a marked atrophy of the right side of the face. No wry-neck was apparent, but on examining the sterno-mastoid carefully, it certainly presented a rigidity that was undoubted when compared with the muscle on the left side. The facial atrophy was as marked as I ever saw it in any case of thoroughly declared congenital torticollis, yet the sterno-mastoid was so slightly affected that the abnormality was only apparent when carefully looked for; it was too slight to interfere with movement or require treatment.

In the state of the sterno-mastoid itself is to be found the means of separating congenital wry-neck from the other condition found in infants, to which I have above referred, and which somewhat resembles it, and which, according to Stromeyer, is the precursor of it.

The sterno-mastoid in congenital wry-neck is found shortened, hard, cord-like, and less thick than that on the left side. It starts out under the skin, especially at its sternal and clavicular attachments. On manipulation it does not at all give the impression of a muscle in active contraction, but rather of one shrunken and stiffened from some cause, or which, having ceased to grow at the same rate as the rest of the cervical tissues, has now become unduly strained from the increasing length of the neck; and further, as a result of this, has kept the right ear inclined towards the same shoulder, with advancement of it also in front of the

lateral median plane of the body. The horizontal level of the eyes is consequently not maintained, the left eye being much the higher, but this is brought about also by the facial hemiatrophy of the affected side. No deposit or local thickening can be discovered amongst its fibres, its relative proportions are undisturbed; but in the other malady, which, as I have said, is thought by some to be the precursor of wry-neck, a notable thickening in the muscle is the thing which first draws attention. It is too marked to be overlooked by the most superficial observer.

The following is a case of this kind, which is quoted as a contrast to those given later on as examples of congenital wry-neck:

Annie P—, æt. 3 weeks, was an out-patient under my care in May, 1882, at Guy's Hospital. The mother noticed, a week after the child's birth, a lump in the right sterno-mastoid, and stated that there had always been a tendency for the milk "to be returned through the child's nose." The palate was normal, and there was nothing in the patient nor in her history to point to congenital syphilis. The left sterno-mastoid was normal, but the right one was cord-like, but not completely rigid. In nearly the middle of the muscle was an oval hard deposit, fairly circumscribed yet not encapsuled; it measured 1 × ¾ inches, and was very hard. All the movements of the neck were perfect, and there was no torticollis.

Hyd. c̄ Cretâ gr. ½, twice a day, was ordered, and in five weeks the lump had disappeared, and the muscle appeared to be perfectly normal in size and consistency. (It is to be regretted that the report of the case made at the time contained no reference to the nature of the labour when the child was born, whether difficult or not.)

Another case of the same kind is recorded by Dr. Frederick Taylor,[1] and it is especially valuable as a microscopic examination was made of the affected muscle. The leading points in his case are as follows:

Jas. C—, æt. 4 weeks, was brought to Guy's Hospital with congenital syphilis (the rash being well marked), and with a lump in the sterno-mastoid of the *left* side. The mother had noticed it two weeks after birth. It had not altered

[1] 'Proceedings of the Pathological Society,' vol. xxiv.

from the time it was first seen. The lump is described as an elongated mass in the lower part of the cleido-mastoid, adherent to the skin, and painless. All the movements were normal, and there was no wry-neck.

The child was treated with Hyd. c̄ Cretâ internally, and Tr. Iodi locally; but in six weeks it died from an attack of pneumonia.

An examination of the muscle showed it to be hard and indurated, and enlarged, as stated above, at its lower part. The cleido-mastoid was thickened from its clavicular attachment to where it joined the sterno-mastoid, and there it became thin and fibrous. The lower two inches of the sterno-mastoid were thin and fibrous also.

A microscopic examination showed the lump to be mainly fibrous, the whole muscle presenting a great admixture and excess of white fibrous tissue.

Beyond the fact that it occurred in a syphilitic child there was nothing in the condition of the muscle suggestive of syphilis as a cause of the disease.

It should be here borne in mind, in view of the argument to follow, that in each case was the opposite side of the neck affected—that in neither case was the muscle shortened, nor was the position nor the movement of the head other than natural.

The view usually held with regard to the origin of congenital torticollis is that of Stromeyer, who ascribed it to the effect of tension on the cervical tissues as the result of a breech presentation at birth. Hueter,[1] following Stromeyer, describes in these cases, as an early stage, a local muscle hypertrophy or callus; and adds, to account for the wry-neck, that gradual nutritive shortening supervenes, affecting for the most part the sternal portion of the muscle; but that both heads may be equally affected. If this were the explanation of the origin of congenital torticollis it seems strange that we do not get in every instance an account of the swelling in the muscle early in life; yet as far as my cases go such is not the case. The swelling is so marked in the affection of the sterno-mastoid in which it exists that it seems impossible it could have been overlooked by the mother,

[1] 'Grundriss de Chirurgie,' ii, pp. 359, *et seq.*

had it ever existed; and as further showing that some other cause for torticollis must be sought for than injury to the muscle from a difficult labour, in my cases not one was a breech case, nor was the labour in any instance of peculiar difficulty,—in one it was all but instantaneous, and over before assistance could be fetched.

No doubt the microscopic evidence in Dr. Taylor's case would favour the view of some rent of the muscle, repaired by fibrous tissue; and the fact that in two cases one is affected on the right side and the other on the left (although in neither is there any record of a breech presentation) suggests that the cause producing the thickened muscle had some extraneous or chance element about it.

Again, in these cases of hard and thickened sterno-mastoid, there is, as a matter of fact, at the time the thickening exists, no shortening of the muscle, and therefore no torticollis; though of course the possibility of this condition occurring later cannot be denied, but at present it is not proved.

Congenital torticollis further being an affection of the right side hardly suggests a traumatic origin for it: on the supposition that traction at birth caused it, in a breech presentation, it might be expected to occur sometimes on the left side; and in the two cases quoted of "muscular callus" of the sterno-mastoid, one was on the right, the other on the left: but that the injury of the sterno-mastoid thus produced results in wry-neck seems to me, from what evidence I have been able to collect, a gratuitous assumption; and further, allowing for argument's sake that a difficult labour (breech presentation) caused congenital wry-neck, this malady ought to affect the left rather than the right side; it being the rule in obstetrics not only to follow "Carus's curve" in extracting the head, but to incline the trunk of the child upwards—*i. e.* to the mother's right; and therefore, the child's occiput being posterior, the strain, if any, falls on the left sterno-mastoid, and not upon the right one.

Whilst, therefore, partial rupture of the muscle may be the cause of "muscle callus" in such cases as the two narrated, there is no proof that wry-neck begins of necessity in such a condition, nor in connection with labour at all;

and even did congenital torticollis cases give uniformly a history of breech presentation, with injury to the muscle, there still would be unexplained the facial hemiatrophy to which I will now refer.

This hemiatrophy of the face seems to be rather different from that form of the affection with which physicians are acquainted,[1] and which has been observed to follow injury of the face, affections of the sympathetic in the neck, and certain exanthemata. These cases, for the most part, show not merely a diminution in size of the side affected, but also a disproportion of parts amounting to deformity, with some alteration in texture and colour of the skin and hair. It is very difficult on physiological grounds to see one's way to accepting a peripheral agency as productive of the hemiatrophy; and one case given by Payne,[2] to which reference will be presently made, although reported apparently with the idea that its origin was in some peripheral disturbance of nutrition, appears to me strongly to argue in favour of the view I shall advance as to the cause of congenital torticollis.

Dr. Hale White, in an exhaustive and studious paper on the Pathology of the Human Sympathetic,[3] discards the idea of injury to this nerve being looked upon as a cause for hemiatrophy of the face, and criticises the cases that have been quoted in support of it. Falling back on physiological grounds, he rightly points out that destructive injury to the sympathetic, if it produced any nutritive change in the tissues, should cause hypertrophy, through vaso-dilatation; and that irritation of it, giving rise to vaso-constriction, ought to produce atrophy. Experiments on animals bear out at least the former of these conclusions; whilst destruction of the human sympathetic by tumours or aneurysm does not, as a clinical fact, cause any alteration in the growth of the face on the affected side. Dr. Payne, in his remarks upon his case, comes to the same negative conclusion in regard to the sympathetic. His case was that of a man forty-two years of age, but the disease first appeared at

[1] Gowers, 'Nervous System,' p. 804, *et seq.*

[2] 'Pathological Society's Transactions,' 1881, vol. xxxii.

[3] 'Guy's Hospital Reports,' 1889, p. 85.

eight years of age. The sequence of events in the child was measles, exanthematous muco-periostitis, and alveolar periostitis (of the left lower jaw), loss of teeth, followed by a large inflamed gland under the jaw—a chain of events very commonly to be followed in surgical practice. Then appeared the hemiatrophy of the face. Now may not this last have been due to some intra-cranial condition about the nucleus of the fifth pair of nerves, of the nature of a local encephalitis induced by measles, from which we have evidence that children suffer? If this is the right reading of his case, it assimilates it to mine of congenital torticollis; though in his case there was alteration—a premature aging—of the tissues, besides arrest of their growth; which latter is the only change in the facial hemiatrophy of congenital torticollis. What little is known of the influence of the fifth pair of nerves on nutrition gives weight to the suggestion that somehow or other that nerve is to blame for facial hemiatrophy when accompanied with degeneration of tissue.

Eulenberg, to explain the hemiatrophy in torticollis, advanced the idea that the position of the neck due to the shortened sterno-mastoid sufficed to interfere with the vessels and nerves ascending to the head, and so brought about an arrest of nutrition. The probability of even a complete inclination of the head on the shoulder producing a material change in the vascular supply to the head above is contrary to general experience; it would surely require a greater degree of lateral flexion of the neck than could be possibly produced to bring about such a result; extreme flexion, such as is not there possible, is necessary in other parts of the body to retard the blood-flow. The flexion of the fœtal chin on the sternum is far greater than that produced by a short sterno-mastoid; and were Eulenberg's suggestion even probably true, we ought to see a double facial atrophy as the rule after a normal pregnancy and delivery.

The anastomosis of the blood-vessels, too, in the head and neck is so perfect, that were even the vessels occluded on one side, experience teaches that those on the other would be ample for the supply of blood; whilst, further, after ligature of the carotid, no chronic atrophy of the tissues supplied by it is ever seen. The brain on the right side should in

the same persons suffer atrophy; and we might expect to discover it in some muscle failure on the left side of the cheek; but such is not the case: I know of no evidence of deficiency of power of the right side of the brain in congenital torticollis.

Gowers[1] refers to Busch's remark that the inclination of the fœtal head naturally to the right *in utero* might bring about a shortening of the right sterno-mastoid. He says in a foot-note, "It [wry-neck] is much more frequent on the right side, which gives support to the theory, as Busch points out, since in 70 cases out of 100 the head is in the 'first position,' which entails an inclination such as would permit the right sterno-mastoid to shorten."

In both these views the shortened muscle and flexed neck are employed to explain the ill-development of the face, and on that ground alone I should dispute their etiological value, since I believe, on grounds to be presently stated, that the affections of the face and muscle do not stand to each other as effect and cause, but that both are the expressions of some central mischief, and that if one may be said to precede the other, it is the hemiatrophy of the face that precedes the shortened muscle, and not the reverse.

The view that has always seemed to me to offer the most reasonable explanation of these cases—and it is one that each case impresses more deeply upon me—is that the pre-existing circumstances of which the deformity of congenital wry-neck is the immediate consequence are similar to, if not pathologically identical with, those that bring about an attack of infantile paralysis and its later complications. Contrast a case of talipes equino-varus with malnutrition of the limb below the knee with one of congenital torticollis: in the former is seen malposition of parts relative to one another; the calf-muscles hard and "contractured," and the inability to flex the ankle-joint in consequence; a badly nourished condition of the soft parts, and often even of the bones, as can be shown by a measurement taken from the knee downwards, and by comparing an outline of the tread of the affected foot with that of the other side. The limb also is cold, and passively congested as a rule. Now all these condi-

[1] Loc. cit.

tions are precisely reproduced in congenital torticollis, unless we except the coldness of the limb; but even this, I have reason to believe, is likely to have been present at the onset; but where it does occur it very rapidly disappears, owing, no doubt, to the perfect circulatory arrangements of the head and neck.

The disease of the central nervous system (acute anterior polio-myelitis) which gives rise to the condition easily recognised as infantile paralysis is not confined to the anterior horns of the spinal cord; the cases that come before a surgeon in the ordinary course of practice—the bulk of such cases being some deformity and paralysis below the left knee—no doubt do arise as such; but in some the patients give a very clear account of a fit, perhaps with convulsions, after which power in the limb has been lost. Such would not be a likely history were the malady confined to a small area in the spinal cord, and yet any brain condition in early life, analogous to anterior polio-myelitis, can be hardly considered as generally recognised. That such a condition does exist in very early life there is no doubt; one well-marked case came under my notice many years ago.

A little girl aged nearly two was left by her mother lying down one afternoon for three hours, and on her return to the child, about 5 p.m., she was struck with the coldness of the left side of its face and arm, and on taking her to the light saw the left half of the face, the arm and leg pallid, and, as she expressed it, like marble. The arm and leg were quite powerless. The child was then perfectly sensible, and the mother did not know whether the child had had a fit or been insensible. I saw the child a few days later, on account of dark, blood-stained bullæ that appeared over the front of the left leg below the knee. Besides this condition I observed the leg to be paralysed and congested, with the bluish congestion rings often observed when a limb in infantile paralysis is very cold. The arm had nearly recovered, and there was nothing then amiss with the face. From the appearance of the skin around the bullæ I thought it likely that gangrene of the skin would follow; and this surmise proved correct, as I long had to treat the child for the resulting sores. As weeks passed the left leg below the

knee wasted, but the growth of the bones was normal, and talipes developed. For over two years the child was under observation, and treated with the interrupted constant (galvanic) current, together with suitable surgical means for the talipes; she made a perfect recovery, and no shortening remained.

Such a case, had I not seen it in its early stage, would in due course have come under notice as an ordinary one of infantile paralysis, and manifesting itself in the most common place, that is, below the knee on the left side; and the cause would have been regarded solely as spinal. That this was a brain affection there can be no doubt; and, indeed, the great frequency of infantile paralysis showing itself in the left leg suggests, for some reason, a predilection of the right brain to early local disease—that is, assuming that more of the cases of infantile paralysis in the left leg begin as my case did in a brain lesion rather than in the spinal cord. The case is a perfect instance of a nutritive hemiplegia; and the sequelæ are just what one would have expected, the nutrition nerve-centre being injured. Two other cases of the same affection I take shortly from my out-patient note-book.

1. Charles R—, æt. 2½, out-patient at Guy's Hospital, April 28th, 1879. Was quite well till eight months old; then one night he cried continuously; the next morning he appeared to the mother completely paralysed, but in a week had recovered power except in the right leg; he never had had "fits," but had suffered from spasmodic croup. He was a pale, pasty, rickety child, with well-marked infantile paralysis of the right leg below the knee; it was wasted, especially the calf of the leg, and very cold; the ankle was fully flexed, forming talipes calcaneus. The muscles of the left leg also were to all appearances wasted, though there was no paralysis; he had walked after a fashion from eight months of age, and had not put the right foot sole down to the ground, always walking on the heel.

Under the galvanic current, and surgical treatment for the talipes, he soon began to improve.

This case, from its history, must have had a cerebral

rather than a spinal origin, and the onset was evidently acute. It is interesting, too, as the resulting paralysis remained more or less permanent in the *right* leg.

2. Alfred S—, æt. 5, out-patient at Guy's Hospital, March 6th, 1882. He was first under observation (at my out-patients) when three weeks old for infantile paralysis of the left side and left talipes varus. The talipes was simply treated by restoration of position with strapping. The case was then lost sight of until 1882. The condition then (æt. 5) was that the right foot, beyond being weaker than the other, was well; the child walked with hardly any eversion, and had done so for the last three years.

My notes continue thus:—"Last Friday (*i. e.* February 28th, 1882) the child had a spasmodic attack, with twitching of the left side and with left strabismus; she also had delusions, thinking there were rats in her cot and taps of water constantly running; she also has night terrors. The hands and face (left) seem now quite right. The child is not at all stupid-looking, but is languid." ℞ Hydrobromic acid with steel.

March 20th.—Much improved; no delusion, terrors, or twitchings.

April 3rd.—Still better, but has very bad headaches at times.

This last spasmodic attack must have had a cerebral origin in the right brain: the case agrees with those of acute polio-encephalitis; and though no paralysis followed this attack, it is highly suggestive that the original infantile paralysis of the left leg had a cerebral rather than a spinal origin; and whatever, at that time, may have been the exact intra-cranial lesion, it seems to have left some permanent mark behind it, and so to have lighted up afresh years after when the attacks of unilateral spasm and severe headache appeared.

Under the name of primary acute encephalitis, Strümpell[1] describes cases of which I believe those I have quoted are examples. It occurs usually before the fifth year, and is generally accompanied by feverishness, vomiting, headache

[1] 'Deutsches Archiv für klin. Med.,' Bd. xlvii, H. 1 and 2.

and general bodily disturbance. Convulsions and coma follow in a few hours or after the lapse of days, and then on recovery a more or less limited paralysis remains.

Ashby, in the 'British Medical Journal' of last year,[1] draws attention to the great similarity in sequelæ between acute anterior polio-myelitis, and the acute local encephalitis or acute polio-encephalitis described by Strümpell; and remarks to the effect that every surgeon must be able to bring to mind cases in all points resembling infantile paralysis as commonly recognised (*i. e.* acute anterior polio-myelitis), and yet in which the history clearly points to some primary brain lesion, and not to one of the spinal cord. To this remark I may add that at times local signs are left showing that parts higher than the spinal cord must have been affected originally, even where the history given by the parents may seem to contradict this view. With such evidence of the possibility of local intra-cranial lesions early in life and not endangering it, and bearing in mind the exact similarity between congenital torticollis and a case of infantile paralysis with talipes equinus, the conclusion is, to my mind, all but inevitable that "caput obstipum" with its contractured sterno-mastoid, wasting of the facial muscles and soft parts, and even of the bones, forms an exact copy of talipes equinus with contractured calf-muscles, ill-nourished soft parts, and in many instances shortened bones, and must have a similar origin. The low local temperature which is so characteristic of and so persistent in infantile paralysis is certainly absent when we see these cases of congenital wry-neck; but it is only after the disease has existed some time that the child is brought under observation; and there must elapse a long period of aftergrowth, so that the conditions productive of the characteristic deformity, for which the child is brought, may have time to act. Even if, however, the case were seen quite early and could be recognised, I should expect the face to have its normal temperature; for in the case of "nutritive hemiplegia" that I have quoted, the face was perfectly natural a few days after the attack, and the arm nearly so, the leg below the knee alone remaining chronically affected.

[1] February 8th, 1890.

Of course, to clench this argument, my case should have developed a torticollis, or a torticollis case should be forthcoming in which the leg on the right side showed infantile paralysis; but I have not yet been fortunate enough to see one, though doubtless one such will be eventually recorded.

My argument is not invalidated by the fact that congenital torticollis cases do not show evidence of infantile paralysis in the extremities; for we know that local and isolated spots in the brain may be picked out by acute polio-encephalitis.

Instead of following Eulenberg in attributing the facial hemiatrophy to the position caused by the affected muscle, I regard the facial change as, if not coincident with, then earlier than the rigidity of the muscle. I have already mentioned a case of marked atrophy in a girl, with scarcely anything wrong with the sterno-mastoid; and this fact, with the one that right facial hemiatrophy can be often seen when looked for without any wry-neck at all, seems to me to point to both affections having a common origin; and where one exists more markedly than the other it is the face that is the more commonly affected alone. Where the muscle is affected the face always is so as well.

Diagnosis.—The diagnosis of congenital torticollis is not difficult, for it is always accompanied by hemiatrophy of the face; so that the conjunction of this condition with a rigid and short sterno-mastoid, both on the right side, will distinguish the case at once. Many other forms of wry-neck exist in children, but for them some cause may be found, such as spinal disease; or they may not be really affections of the sterno-mastoid, but the oblique position of the head is due to spasm of some other muscle, as the complexus, or to spinal disease, or to some local inflammatory swelling, as cervical spinal abscess, or glandular disease and phlegmon. The affection of the sterno-mastoid seen in infancy, and characterised by a local swelling in and amongst the fibres, whatever its origin may be, is easily distinguished from congenital torticollis, inasmuch as it is accompanied by no wry-neck nor impairment of movement whatever. Whether it is likely to eventually give rise to torticollis has been discussed.

Treatment.—The treatment of these cases is now a settled

matter, and may be found in any surgical text-book; only a few points need reference here.

The employment of any mechanical apparatus to correct the position of the head prior to tenotomy is useless; but after the division of the muscle, as after tenotomy of the tendo Achillis for talipes equinus, extension is necessary; and it is best if the extension is elastic and acting continuously, at least during the day. Restoration to the correct position should be begun the day after the operation; unless, as in one of my cases, severe hæmorrhage should have occurred, when the delay of a day or two is advisable.

It must not be forgotten that though the sterno-mastoid is the cause of the torticollis, it is sometimes aided by the contracted state of the platysma. Such a cause as contraction of the platysma, pure and simple, may exist alone and produce a form of wry-neck: a case of this is given by Gooch and Dieffenbach;[1] but where it exists in congenital wry-neck it seems to be a consequence, and not a cause. In one of my cases it slightly complicated the operation, the fibres of the platysma having to be divided before the head could be restored to its normal position. If in operating the sterno-mastoid be first divided, then the cleido-mastoid, the tenotome can be still further inserted parallel with the clavicle and through the same skin-puncture, and thus be made to divide the platysma fibres if they are contracted. Until the sterno-mastoid has been completely severed it is not possible to say whether the platysma is at fault also or not.

A good form of elastic apparatus, and very simple, is a door-spring of rubber attached above to a webbing head-band laced round the crown of the head, and below to a stout hook sewn on to a pair of stays, temporarily worn for the purpose: the rubber spring has lengths of tape at its ends, whereby it is fastened, and by means of which the tension can be regulated. The attachment above can be made over the left mastoid, and the spring brought down in front of the left shoulder to the hook on the stays; or, better, it can be brought from the right temple over the right shoulder, and made fast behind to the hook, which in this case is sewn on at a spot corresponding to the lower part of

[1] Quoted by Hueter, loc. cit., p. 360.

the left blade-bone. The position of the head should be looking somewhat upwards and to the right of the middle line of the body.

This tension arrangement can only be worn during the day, and if this is done for two or three months the treatment is over.

When first released by tenotomy the muscle still feels firm and cord-like; but in a few weeks it regains its normal consistency, though it may remain rather thinner than that on the other side.

The full treatment adopted in the parallel cases of talipes resulting from infantile paralysis is not applicable in congenital torticollis. The use of the interrupted constant current in the former is of course most effectual on the restoration of the wasted muscles, provided fatty degeneration has not taken place, but it fails in restoring the length of the bones, or in enabling them to catch up those of the other leg in their growth; and since in congenital torticollis it is not the wasting of the muscles of the face that causes the asymmetry, but impaired growth of the bones, and of the cartilage and other tissues of the external ear, we should gain nothing by the employment of electricity; and, indeed, the muscles of the face, though they may be smaller on the affected side, do not seem to have lost their power to any visible degree.

Prognosis.—As far as the neck is concerned cure is certain; but, in spite of Eulenberg's statement that division of the sterno-mastoid soon allows the defective side of the face to become of normal size, I have never seen it, nor can I conceive it to be possible: the half-face of course grows *pari passu* with the sound side, as it has done all along, after the first effects of the cause of the hemiatrophy have passed off; but it does not grow at a quicker rate, which it would have to do to catch the other up. In one of my cases, aged eight years, when I operated, the facial hemiatrophy was well marked in a photograph taken three years afterwards. The position and movements of the head were perfectly normal, and the child was in perfect health.

General Summary and Conclusions.

1. Congenital torticollis is characterised by the constant accompaniment of facial hemiatrophy of the same side.

2. It is an affection of the right side (I have met with no case on the left).

3. It is a distinct affection from that seen in infants, of so-called ruptured sterno-mastoid, which is characterised by a local thickening (muscle callus). This latter affection may be on either side, and is ascribed to an accident at birth: it is unaccompanied by torticollis.

4. It is not due to accident affecting the periphery, but to a central (brain) lesion, viz. acute polio-encephalitis.

5. It is the counterpart in the head and neck of infantile paralysis with talipes in the leg.

6. The facial hemiatrophy is not a consequence of the torticollis, but an integral part of the affection, both having a common central origin.

7. The facial hemiatrophy in some cases probably precedes, or at least is much more marked than the torticollis. The former may exist without the latter; but the converse of this does not hold.

8. The facial hemiatrophy, as also the shortening of the sterno-mastoid, is due to arrest of growth, and is not accompanied by tissue degeneration, or by pigmentation as seen in some cases of hemiatrophy of the face which are acquired after infancy and unaccompanied by torticollis.

9. In cases of doubt the slightest obliquity of the countenance may be recognised by looking at the reflection of the patient's face in a mirror.

10. Treatment can only be directed to restoration of the position and movements of the head; in these particulars cure is certain. The facial atrophy is permanent.

The following are the six cases referred to in the foregoing remarks:

Case 1.—Sidney W—, æt. 10, was in Lazarus Ward under my care in October, 1879. Nothing was observed to be wrong with him until he went to school at five years of

age, and then the schoolmaster observed that he always carried his head on one side. For two years after this he was treated with lotions, with a view of correcting the deformity.

When admitted he had characteristic congenital torticollis of the right side, with marked diminution of the right half of the face. The right sterno-mastoid was very tense, and stood out under the skin. The right eye was extremely myopic; he could only read large letters at five inches with it, but could easily read the same at nine feet with the left eye. The right sterno-mastoid was divided in the usual way, and the spring apparatus applied. He perfectly recovered.

Some years after (date uncertain) the lad presented himself again; he was quite well, but the facial asymmetry remained.

Case 2.—Rose F—, æt. 9, was seen October, 1879, with Dr. Seton, of South Kensington. Since an infant wry-neck had been noticed, her face looking over to the left shoulder, and the right side of the face had been observed smaller than the left. The centre of the left pupil is half an inch further from the middle line than that of the right one. The right sterno-mastoid is rigid and short.

In this case the child's grandmother insisted that the deformity had always been present; but the friends among whom the child's life had been passed, she being an orphan, denied its existence till lately. On searching the family album I found, in a photograph of the patient taken at two years of age, the wry-neck and facial deformity were well exhibited.

On October 21st tenotomy was performed, and for three months after the spring apparatus was worn; my note made January 13th, 1880, says, "Patient perfectly well. There is no perceptible difference between the two muscles now, but the face is still unsymmetrical. Has worn spring up to present time."

Case 3.—Catherine H—, æt. 5, December, 1880, seen with Dr. Roberts, of Peckham Rye. Ever since infancy the

mother had thought that the head "seemed on one side." When the child was two years old professional advice was first sought for the deformity; wry-neck was then discovered, but as it was looked upon as possibly symptomatic of some spinal mischief nothing was then done for it.

The child herself is particularly robust and healthy, and there is no evidence of spinal disease. There is right torticollis, the sterno-mastoid being rigid and shortened. The malposition in this case is extreme, the opposite side of the neck bulging prominently, so that the left sterno-mastoid has the appearance of hypertrophy. The facial atrophy is marked; the right external ear is a quarter-inch shorter than the left one, and the vertical measurement between the outer canthus of the eye and the angle of the mouth on the right side is a quarter-inch less than on the left. The pupils are both central and equidistant from the middle line of the face; the child is deaf with the right ear. (This was specially mentioned by the mother; it proved to be due to cerumen, and syringing cured it at once.) There is no want of symmetry inside the mouth to be seen.

I performed tenotomy on December 11th, 1880, and whilst dividing the cleido-mastoid a profuse gush of venous blood occurred; on withdrawing the tenotome and closing the puncture a blood-tumour the size of an orange at once appeared. On squeezing out the blood and again closing the puncture the swelling reappeared; on again evacuating it and putting a compress over the site of the divided muscle all hæmorrhage ceased. The compress was retained *in situ* for two days; it was then removed, and no further complication arose. The spring apparatus was applied, and on March 2nd, 1881, the child was seen perfectly well, the sterno-mastoid being supple and apparently normal. The hemiatrophy of the face was unchanged.

Case 4.—Emily D—, æt. 2½, was seen December, 1889, with Mr. Duke, of Clapham. A month previously the child had been taken to Mr. Duke on account of some small ailment, and he then noticed it was the subject of wry-neck. The parents had not observed the deformity themselves.

An examination revealed right torticollis, the right sterno-

mastoid being short and rigid, the clavicular portion seeming especially hard and cord-like. The right half of the face was much smaller than the left, so that the loss of the horizontal level of the eyes was the first thing apparent.

On December 7th I performed tenotomy, restoring the head to the normal position at once, and the next day the spring apparatus was applied.

The child made an uninterrupted recovery, but the face remains still unsymmetrical.

Case 5.—Dora De F—, æt. 8, I saw with the late Mr. Bianchi, of Blackfriars, in January, 1886.

The deformity had only just been observed, and was characteristic of congenital wry-neck—rigidity and shortness of the right sterno-mastoid with marked facial atrophy of the same side. The right external ear was a quarter of an inch smaller than the left one. Tenotomy was performed without any difficulty, and the spring apparatus directed to be worn. In three months the child was reported quite well; but a photograph taken of her three years later shows the position of the head to be normal, but the facial asymmetry is as marked as ever. In this case my notes specially remark that the labour was normal and very rapid, being over in a few minutes.

Case 6.—Gilbert W—, æt. 7, was seen in October, 1890, with Mr. Benjamin Rix, of Tunbridge Wells.

Attention had only just been drawn to the deformity, nor were the parents convinced of its having existed a long time until shown it in an early photograph. When examined the case presented all the characteristics of congenital torticollis of the right side, together with very apparent right facial atrophy, the vertical measurement on the right side from the inner canthus of the eye being three and a quarter inches, against three and a half inches on the left side; but both ears were of equal size.

I performed tenotomy on October 27th; but after the complete division of the sterno-mastoid there were seen several subcutaneous bands over a distance of fully an inch beyond the divided muscle, which prevented restoration of

the head to the middle line. They were clearly platysma fibres, and their division by the tenotome was necessary before the head could be put straight. There was no bleeding beyond what is seen in ordinary tenotomy punctures.

The following day Mr. Rix applied the spring apparatus, and retained it in action for three months. He then reported to me that the child was perfectly well, the muscle soft and apparently of normal suppleness, and all the movements of the neck natural, the facial asymmetry remaining as at first.

In this case also there was nothing exceptional about the labour when the patient was born.

NOTE ON THE URINE IN A CASE OF PHOSPHORUS POISONING.

By ERNEST H. STARLING, M.D.,

AND

F. GOWLAND HOPKINS, B.Sc.

Published accounts of the exact composition of the urine in cases of phosphorus poisoning not being numerous, we have thought it worth while to give an account of this case, though, to our regret, we were unable to obtain more than one specimen of the urine (passed in twelve hours), and so could not follow the case throughout. We are indebted to Dr. Goodhart for kind permission to publish this case.

Abstract of Report of Case.

Alice G—, æt. 18, admitted into Guy's Hospital, January 23rd, 1891.

On January 21st, the patient ate by mistake a slice of bread and butter which had been thickly covered with phosphorus paste. Next day (January 22nd) she complained of pain in abdomen and headache, and on the 23rd she vomited twice.

On admission.—Pains in abdomen and head, considerable thirst, tongue thickly furred. Bowels have not acted for

three days. Liver enlarged, hepatic dulness extending two inches below margin of ribs. Urine sp. gr. 1046. Urea 2·8 per cent.; no albumen nor blood.

On the 25th she felt better. The liver dulness was less (reaching only one inch below ribs).

On the 27th the spleen was perceptibly enlarged, and the patient slightly jaundiced.

At 2 a.m. on the 28th she suddenly became worse, with dyspnœa, the pulse becoming almost imperceptible. She gradually sank, and died from failure of heart and respiration at 9.15 p.m.

In the twelve hours from 6 p.m. on the 27th to 6 a.m. on the 28th she passed 16 oz. of urine, which were given to us to examine. A few hours later she passed some urine with a motion, and no more till her death.

Post-mortem.—Liver 66 oz., fatty. No bile in gall-bladder. Spleen 7 oz. Petechiæ found in various parts of the body. Kidneys large (14 oz.) and mottled. Bladder empty.

Analysis of the Urine.

Amount passed in twelve hours from 6 p.m., January 27th, to 6 a.m., January 28th, was 528 c.c.

	Parts in 100 of urine.	Total quantity.
Urea	2·5	13·2 grammes.
Ammonia	0·18	0·95 „
Total nitrogen	1·344	7·096 „

Nitrogen as urea . . 1·17 per cent.
Nitrogen as ammonia . ·148 „

Ratio of nitrogen in urea to nitrogen in ammonia = 7·9 : 1.
No leucin or tyrosin.
Total sulphur . 0·240 per cent.

Oxidised sulphur . 0·122 „ { ·116 as sulphates. ·006 as conjugated (æthereal) sulphates.

Ratio of unoxidised to oxidised sulphur = ·97 : 1.

The urea was estimated by the hypobromite method.

The total nitrogen was determined by Kjeldahl's, and the ammonia by Schloesing's method. The sulphur determinations were made in the way indicated by Bunge ('Physiological Chemistry,' p. 362).

In examining for leucin and tyrosin, 100 c.c. of the urine were precipitated with lead acetate, filtered, and the filtrate treated with H_2S to precipitate the excess of lead. This was filtered off, and the clear fluid thus obtained evaporated to a syrupy consistence on the water-bath. On examination of this residue a few bodies were seen under a microscope, which we took for "leucin cones," but there was no trace of tyrosin to be found either in this way or by chemical means. The quantity of the "leucin," if present, was too small to be subjected to chemical tests.

The urea and ammonia together are very nearly enough to account for the total nitrogen.

It will be seen on looking over the above analysis that the urine is very nearly normal. Yet the slight deviations it presents from the normal are interesting. These chiefly concern the ratio of the nitrogen excreted as urea to that excreted as ammonia, and the ratio of the unoxidised to oxidised sulphur.

In health the amount of nitrogen excreted in the form of ammonia varies between 0·3 and 1·2 grammes a day (Neubauer, 'Journ. f. prakt. Chem.,' 64, 177). Taking an average, we may say that the proportion of N (as NH_3) to N (as urea) is about 1 : 70.

We have been unable to find any account of the relative amount of N excreted as NH_3 in phosphorus poisoning. In some other diseases of the liver, such as cirrhosis, it is said to be increased fourfold (Stadelmann, quoted by Bunge, p. 327 ; see, however, Fawitsky, 'Centralb. f. Pathologie,' vol. i, p. 313).

In this case the proportion was 1 : 7, showing a large relative amount of N as NH_3. This is what one would expect. The amount of ammonia excreted seems to be largely conditioned by the acidity or alkalescence of the plasma.

If a large amount of acid be administered to a starving animal we find immediately a large increase in the ammonia of the urine. The acids rob the tissues of the ammonia

radical which otherwise would have left the body in the form of urea.

Meyer[1] has shown that in cases of phosphorus poisoning there is a marked decrease in the amount of CO_2 to be extracted from the blood.

The amount of CO_2 in the blood is chiefly conditioned by the alkalinity of the plasma, and this decrease of CO_2 found by Meyer points to an increased acidity, or rather a diminished alkalescence of the plasma. In fact, in phosphorus poisoning we have a condition of the plasma similar to that one finds in cases of diabetic coma. Hence one would expect to find in these cases, just as after administration of acids, an increase in the relative amount of nitrogen excreted as ammonia.

The other index to the proteid disintegration in the body is the sulphur.

Normally the ratio of unoxidised (bodies like cystin) to oxidised sulphur in man is about 1 : 5 (Salkowski, quoted by Neubauer and Vogel).

In our case the ratio was 0·97 : 1, pointing to a large increase in the excretion of the unoxidised compounds of sulphur. This increase was also noticed by Goldmann and Baumann in some cases of phosphorus poisoning ('Zeitsch. f. phys. Chem.,' vol. xii, p. 254).

Blendermann has also noticed increase of the conjugated sulphuric acids in a case of phosphorus poisoning.

In our case the proportion was rather below normal, the ratio of sulphur in the conjugated acids to the sulphur as normal sulphates being about 1 : 20. Normally, according to Van der Velden, the ratio is 1 : 10.

The urine was perfectly free from albumen, but there was a slight deposit of squamous epithelial cells, which would account for some of the unoxidised sulphur.

[1] Hans Meyer, 'Arch. f. Exp. Path. u. Pharm.,' xiv, p. 313.

A CASE OF COMPRESSION PARAPLEGIA.

By E. W. GOODALL, M.D.,

WITH AN ACCOUNT OF THE MICROSCOPICAL EXAMINATION OF THE CORD.

By E. GOODALL, M.D.

Edward D—, æt. 62, admitted into Guy's Hospital for paralysis of the legs on December 2nd, 1889, under the care of Dr. Goodhart, to whose kindness we are indebted for permission to publish the case.

Previous medical history.—Patient was born and has always lived in London. Worked as a tailor until two years ago. He has taken alcohol pretty freely. Always had fairly good health until two years ago; having, however, suffered from bronchitis and pleurisy. Two years ago he was taken ill with severe coughing, and he lost flesh rapidly. He got worse, and six months ago he had to keep to his bed. A fortnight ago he lost the use of his lower extremities, and was also unable to sit up in a chair or to feed himself. At the same time he had retention of urine, which had to be drawn off with a catheter. He has had to have it drawn off once since.

Condition on admission.—The patient is much emaciated; he is very helpless; was carried up into the ward. There is loss of power, complete, in the lower extremities (the notes do not state their position, but from a note made on December 7th they were apparently extended); no loss of common sensation, but sensation of pain is impaired in the lower extremities. Epigastric, abdominal, and cremasteric reflexes

absent; plantar reflex present on both sides; knee-jerks absent; no ankle-clonus. The muscles of the lower extremities are wasted, and also those of the upper, but the grasp of each hand is fairly firm. There is prominence of the spine at about the fourth dorsal vertebra. There are blisters on the feet caused by the application of hot-water bottles, and a bedsore over the sacrum.

Patient's face has a vacant expression, and he lies in bed in an apathetic way; he is inclined occasionally to wander in his talk. He can pass his urine, which is acid, of a sp. gr. 1020, and loaded with amorphous urates. There is no retention or dribbling away of the urine. He is troubled with a cough, and expectorates thick muco-purulent sputum; there are well-marked physical signs of a cavity in the upper lobe of the left lung. The chest is flattened on both sides; the lower spaces on each side sink in on inspiration; the breathing is chiefly diaphragmatic. There is a loud systolic murmur to be heard all over the precordial area, and, in fact, beyond that limit, though less loudly; it is loudest over the aortic area. No enlargement of the heart to be made out. Pulse 96. (In the following report no further mention will be made of the lung and heart symptoms; they altered but little up to the patient's death, and have no bearing on the neurological aspect of the case.)

December 7th.—Patient is beginning to have more power in his legs; he lies in bed with his legs semi-flexed instead of extended as before. When asked to do so, he can draw up his legs a little.

12th.—Can now move his toes a little. There is a sore on the right calf, and the blisters on the feet do not heal.

13th.—Passes his water with difficulty, and has partial retention; bowels have been constipated; glycerine enema ordered.

17th.—Patient complains of twitchings in the legs, and pains running up and down from the legs to the back; he does not wander so much in his talk. There is a little pus in the urine.

20th.—The sore on the calf and the blisters on the heel are now sloughing.

27th to January 2nd.—Patient is troubled with diarrhœa;

the motions, which are liquid, are passed under him. This yielded to treatment.

30th.—Intercostal muscles move better.

January 12th.—Very little change in patient's condition.

15th.—Complains of losing power in his right arm, but on examination the grasp of the right hand is fairly good when compared with the left.

17th.—Patient has fair tactile sensation in his legs, but sensation to pain is diminished ; he has no control over his sphincter ani, and passes his motions into the bed. He complains of passing water only in drops. His legs are not more wasted than on admission ; the muscles are very flabby.

25th.—Breathing still chiefly abdominal ; lower ribs move a little.

26th.—Diarrhœa again, but only lasting for a day.

February 13th.—There is now a bedsore over each trochanter major.

19th.—Has passed his urine involuntarily for the last few days. There is now constipation. He lies in bed with his legs drawn up ; there is no voluntary motion in them. His hands and arms are about the same as when he first came in.

26th.—Patient is lying on his left side with his left knee flexed at a right angle, and his right at an angle of 130°. The hamstring muscles are slightly contracted, but certainly not enough to prevent the getting of a knee-jerk (if one were present). The feet are at a right angle with the legs. The toes of the left foot are a little hyper-extended, especially the great toe. Every now and then there is an irregular movement of the muscles of the thighs and legs, lasting for a few seconds ; sometimes these movements are set up by pinching the skin or tapping the leg, sometimes they occur by themselves. Idio-muscular contractions (of both varieties)[1] can be set up by tapping the muscles of the thighs and legs. These contractions can also be obtained in other places ; in the upper extremities, for example. There is absolute loss of voluntary power in the lower extremities. There is absolute loss of sensation to touch, pain, and temperature in the

[1] See paper by MM. Féré and Lamy, 'Archives de Physiol.,' Juillet, 1889, p. 570.

feet, legs, and thighs. Plantar reflexes are present in each leg, and vary; sometimes only the toes move, sometimes the whole leg, and sometimes both legs. No knee-jerk can be obtained, and there is no ankle-clonus on either side. The loss of sensation extends up on the trunk to the epigastric angle. There are irregular involuntary movements of the muscles of the back and pelvis.

The abdominal and epigastric reflexes are absent (cremasteric not tried for). Respiration is almost entirely abdominal, there being no movement of the muscles of the six lower spaces, and only slight movement in the muscles of the upper spaces. There is eversion of the left lower ribs and bulging of the abdomen there. Sensation is unimpaired above the level of the epigastric angle, and at this level there seems to be a little hyperæsthesia.

Patient has impaired power in his arms, but not more so than can be accounted for by his general weak condition. Elbow-jerk present on each side, stronger on the right. No wrist-jerks. Patient passes his motions under him involuntarily, and his urine dribbles from him. There is a bedsore over each heel and each trochanter major. Mind perfectly clear.

From this time until his death the patient never regained power in his lower extremities. On the occasions on which I myself examined him, he had total loss of tactile and pain sensation below the epigastric angle and the margins of the ribs. The thighs became gradually more flexed upon the abdomen, and the legs upon the thighs; but up to about June 17th the muscles were never so rigid as to prevent the passive extension of the joints through several degrees, or to preclude the possibility of obtaining a knee-jerk; but I could never obtain a knee-jerk or ankle-clonus, nor could any other person who examined the patient. (After June 17th the legs became more rigid in flexion; so that, perhaps, that may have prevented the knee-jerk being elicited. But striking the patella tendon did not give rise to any, even the slightest, contraction of the quadriceps.) The patient's urine used more or less continually to run from him, and a catheter was never required. As regards the bowels there was sometimes constipation and sometimes diarrhœa.

March 11th.—Plantar reflexes present; knee-jerks absent. Patient seems able to feel in the region of the epigastrium, but not in the lower extremities.

22nd.—Diarrhœa.

April 2nd.—Patient continues much about the same; the bedsores are somewhat better, and he says his appetite has much improved. He has still the same amount of anæsthesia in his lower extremities, which appear to be getting rigid in the flexed position. No diarrhœa now.

9th.—The left leg is now flexed at an acute angle with the thigh, and the right at a right angle. They are rather rigid, but not sufficiently to exclude the possibility of a knee-jerk. There is, however, no knee-jerk. There is loss of sensation below the level of the twelfth dorsal vertebra behind, and the eighth rib in front at the margin of the thorax. The muscles of the thigh contract on being stimulated (idio-muscular contractions).

17th.—The bedsore on the right hip is getting worse. Those over the sacrum and left hip are healing a little; those on the feet are quite well. Patient complains of pain in back.

May 7th.—Anæsthesia and analgesia of lower extremities; knee-jerks absent; plantar reflexes increased; no voluntary movement of limbs.

12th.—The bedsores are all worse.

17th.—Bedsore over left trochanter is deep; small pieces of bone coming away.

June 6th.—In same condition.

14th.—Bedsore larger.

17th.—Legs more flexed, the right heel almost touching the corresponding buttock; the left leg is at an angle of 45° with the left thigh. Toes hyper-extended. Diffused reflex on touching soles of feet, causing not only the same leg to contract, but the other also. (The movement was one of flexion.) During the past few days patient has been complaining of what he calls "twitchings" in the thighs, abdomen, chest, and down the spine. He complained of those up to the time of his death.

July 2nd.—Right thigh and leg now in extreme flexion; the left thigh is flexed to less than a right angle with the

trunk, and the left leg to an angle of 40° with the thigh. Otherwise patient is in same state.

15th.—Urine neutral; slight trace of albumen (for first time); it has been alkaline once or twice before.

18th.—Urine alkaline and very slightly ammoniacal; albumen present. Bedsores have been getting bigger. Patient is worse.

21st.—Has been gradually getting weaker. Has been wandering in his talk at times. Looked collapsed on July 19th; is more so to-day. Died at 2 p.m.

The temperature since April 1st has not been regularly taken; but when it was, it was usually up in the evening, and about normal in the morning.

Post-mortem examination (made twenty-five hours after death).—Extreme emaciation. Large bedsores over each trochanter major; smaller ones on heels. No rigor mortis. As the body lay upon its back the legs inclined over to the left side. Each foot was at a right angle to the leg, the left being somewhat everted and the right inverted. The left leg was flexed at the right angle with the thigh, and the thigh to rather less than a right angle with the trunk. On the right side the flexion was more pronounced, the thigh being flexed on the trunk and the leg on the thigh almost as far as they could be. I tried forcibly to straighten the limbs at the knee and hip joints, but could not do so, being only able to straighten them by a few degrees, especially in regard to the knees. There was no crackling in the joints. The hamstring muscles were much contracted. The position described above is that mostly assumed by the patient during the last weeks of life.

Brain normal to naked eye, 46 oz. On the viscera being removed (from the thorax) a yellowish mass was found opposite the bodies of the fifth and sixth dorsal vertebræ, or rather in connection with them; looking at first sight like growth, but showing on section caseous material. The bodies of these two vertebræ were carious, being soft, and had to a large extent disappeared. Caseating material projected into the vertebral canal compressing the cord, which was much narrowed at this place. The cord felt here rather soft, as it did also above; but below it was firmer. To the naked

eye neither ascending nor descending degenerations were apparent. There was no meningitis, and the membranes were not in the slightest degree adherent to the cord. I slit up the membranes easily with a pair of scissors the whole length of the cord. The anterior crural and sciatic nerves were normal to the naked eye. There was no reason during life to suppose that any neuritis of these nerves existed. There was tubercular disease of both lungs, recent and old, mostly on the left side. There was stenosis of the aortic valve, and the aorta was atheromatous. There were a few ulcers in the cæcum and the ascending colon, and a few submucous hæmorrhages in the bladder.

Microscopical examination of the cord.—The cord was hardened in chrome; first in Müller's fluid, then in 2 per cent., and lastly in 4 per cent. Pot. Bichrom. The greater part of the time it was in the 4 per cent. solution. As after the lapse of a considerable period it proved to be unfit for cutting, certain segments were removed and passed through celloidin. Even after this treatment many of the sections subsequently cut presented a hole with ragged edges behind the posterior commissure, produced in cutting; and, as seems probable, primarily due to the diseased state of the tissue. The celloidin, moreover, prevented the proper action of the stain (anilin blue-black) upon the nerve-fibres in the periphery of the cord; though doubtless the wax in which portions of the cord were subsequently imbedded was partly responsible. Notwithstanding, also, the use of origanum oil the pia became detached in the clearing process in many sections, carrying with it the immediately subjacent cord-substance. Still, a large number of sections suitable for study (to which, however, those just referred to were also submitted) were obtained, 230 being taken from the compressed portion of the cord. Attention was paid to the cutting of the narrowest (most compressed) part, in order that the whole of it might be secured for examination; the sections included this and the adjoining broader—though still compressed—parts above and below. In addition, numerous sections were taken from the cervical enlargement; and the portion of the lumbar enlargement corresponding to the roots of the second, third, and fourth lumbar nerves was cut.

The anterior crural nerves were unfortunately in a twisted and shrivelled state when they came to hand, from lack of stretching and a too prolonged stay in alcohol. No sections worth studying could be obtained from them. But—as will appear later—sections of the roots of the attached lumbar nerves were obtained with the cord-sections. For staining, anilin blue-black and the Weigert-Pal method were employed. Safranin (Adamkiewicz) did not appear to furnish results equal to those of the latter method, and was not consistently used.

The compressed portion; anilin blue-black sections.—On taking a general view of a section under the one-inch objective, the eye is struck by the number of large vessels in the field, appearing transversely and longitudinally cut. Many filled by deeply stained clot can be seen, and all have walls of considerable thickness. The vessels are most abundant in the posterior columns and horns, and in the posterior half of lateral columns. From the appearance of the sections the cord in this region has suffered from a chronic inflammatory process; new tissue—of the nature of connective or intercellular tissue, probably composed of both—stained a deep blue colour, is everywhere prominent, to the exclusion of the nerve element proper. Nerve-fibres can be seen with this power only in the posterior root-zone, and in the part of the lateral column which adjoins the posterior horn. Bands of a deep blue colour (vessels and connective tissue) are to be seen running outward from the horns, especially in the posterior half of the section. The anterior fissure contains a mass of connective tissue with large blood-vessels. There is no definite postero-median septum, the postero-median columns being welded into one piece by hypertrophied blood-vessels and connective tissue. The cornua can be distinguished from the surrounding tissue by their deeper tint, but not by the presence of nerve-cells; the diseased cells are only with difficulty identified. One anterior horn has undergone marked lateral compression, and its blue colour shades off into that of the surrounding tissue. The appearance of the horns leads one to anticipate the discovery of advanced disease with a higher power. The nerve-roots are either very considerably diminished in number, or else have

been thrust out of sight and rendered indistinguishable by the growth of new tissue; mere traces of them are visible. The commissural band between the horns of either side is no longer divisible into two portions; white commissure and grey (with its gelatinous substance) have become fused into one broad band, deeply and diffusely stained, and shading off into the surrounding tissue.

Under the same power the Weigert-Pal sections show a great quantity of unstained (connective) tissue, especially in the anterior columns, the fore-part of the lateral columns, the postero-median columns, and the outer part generally of the cord. Healthy-looking rings (medullated nerve-fibres) are seen about the posterior horns, both on their inner and outer aspects, and in the crossed pyramidal tracts. With the exception of a stray fibre here and there nerve-roots are not visible. Large, numerous, chrome-stained structures, of globular and elongated form, constitute prominent objects against the unstained connective-tissue background. These —which are stained blood-corpuscles and clot—draw attention afresh to the implication of the vessels in the disease.

With the $\frac{1}{4}$-inch objective (anilin specimens) the nerve-cells in all parts of the cornua appear much diseased; they are few in numbers, indistinct, shrunken, rounded, and processless, and most are devoid of nuclei. No spaces are visible round them, as in ordinary specimens. Many show large collections of yellow pigment unstained by the dye. The grey matter is throughout deeply stained, and no proper structure can be made out in it; it has none of the normal spongy appearance, the interlacement of nerve-fibres and the transversely-cut axis-cylinders of medullated nerves being almost entirely absent.

As regards the white matter in these sections from the most diseased part, one sees throughout the field deeply-stained tissue (connective and intercellular) and degenerated nerve-fibres. Many of the latter are much swollen, the medullary substance appearing as an irregular mass, and the majority present a granular appearance, as though the medullated sheath had undergone disintegration. Axis-cylinders are entirely absent from most of these fibres; such cylinders as are present are commonly swollen and poorly

stained. The medullary substance is in many fibres blue-tinted from absorption of dye. Areas occur in which mere traces of nerve-fibres remain. In addition to these appearances, amyloid bodies are met with, though not in large numbers.

The morbid conditions above mentioned prevail throughout the antero-lateral columns. In the anterior column, what appears to be a healthy nerve-fibre is met with here and there—very infrequently. Healthy-looking fibres also occur, and more frequently, in the crossed pyramidal tracts, chiefly at their inner part; also some in the region of the lateral cerebellar tracts, at their inner parts (the outer parts have been somewhat obscured by the imbedding material). In the lateral columns in front of the crossed pyramidal and cerebellar tracts no healthy-looking fibres are visible in some of the sections. In the posterior columns morbid appearances are again prevalent. A healthy-looking fibre is very rare in the postero-median tracts; in the posterior root-zones, however, many fibres with normal appearance occur—more than in any other region of the cord.

Under the same power the Weigert-Pal specimens show that the intra-cornual meshwork of medullated fibres is very poorly formed, in comparison with the meshwork of a healthy dorsal region. Throughout the white matter irregular masses of medullary substance occur, some stained black, others light brown or purplish. These have no unstained centre (corresponding to an axis-cylinder), and the dark rings so abundant in a healthy specimen are rarely seen in these sections. Those which are present are situated mainly in the posterior half of the cord, and immediately external or internal to the posterior horns. In general it may be said that these sections bear out the evidence furnished by anilin ones.

The lumbar enlargement.—To the naked eye this appeared quite normal as regards size and symmetry, in respect alike of the two halves of the cord and their individual parts. The nerve-roots seemed healthy. Degeneration was evident in the crossed pyramidal tracts (the hardened cord is here referred to), but no sign of it was visible elsewhere. Microscopically this degeneration was verified; there was nothing

uncommon about it. No other system-degeneration found. Viewed by transmitted light, each section showed cornua of average size and shape. With regard to the cells in the anterior horns, a great number in each horn showed collections of golden-yellow pigment in unusual amount; a few showed nothing but such pigment, which had failed to take the anilin stain. Other cells—in the minority—were free from yellow pigment. In addition to the pigmentation, in many of the sections the cells were not so numerous, and most of them not so well formed, as those in similar regions of the healthy adult cord. (By defect in formation paucity of processes is particularly meant.) Other sections, however, showed a fair number of well-formed cells. Some showed a marked difference in the number, size, and appearance of the cells on the two sides; whereas on the one side they were numerous, large, well formed, and but slightly pigmented, on the other they were small, few, and much pigmented. (Variations of this kind occur in specimens accounted healthy, and not uncommonly in patients of the age of this man). The intracornual meshwork and the anterior and posterior roots within the cord appeared quite healthy. Many sections showed portions of the roots outside the cord—although celloidin had marred the effects of the stain they appeared to be healthy; where cut transversely, the sections showed numerous nerve-tubules with well-stained axis-cylinders. That every nerve-fibre looked intact cannot be claimed; some fibres showed large and poorly stained axis-cylinders, others none at all. But the impressions made upon the observer were that the great bulk of the fibres was healthy; that there was no obvious disease; that no reason existed for supposing conduction had been interfered with; that considerable allowance was due to the imbedding material.

In the white columns some rather prominent connective-tissue septa were seen, especially in the posterior columns. But, with the exception of those fibres bordering on the periphery of the cord—these had a blurred appearance in all parts, for which an artificial source can with much confidence be assumed—no certainly-diseased nerve-fibres were discovered.

Sections were cut from the *cervical enlargement*, mainly with a view to determine whether or not the failure to take the dye manifested by many cells in the lumbar region was also exhibited by the cells in the cervical region. Sections were stained for an hour and a quarter in anilin, and after this treatment many cells were found to exhibit golden-yellow pigment in considerable amount, much greater than is seen in an ordinary specimen from an adult cord. Moreover, the number of cells present in the anterior horns was small—often smaller on one side than the other; and the cells themselves in many instances were much stunted. These conditions—though less obvious than the corresponding ones in the lumbar region—were still sufficiently pronounced to show that the latter may have been due in part at least to natural causes. Similar appearances (pigmentation, stunted cells) are common in senile cords, the functions of which had been efficiently performed.

There was degeneration of Goll's columns above the compressed portion, and for a short distance of Burdach's also.

Remarks.—The chief point of interest about this case was the continued absence of the knee-jerks. Dr. Bastian, in a paper read before the Royal Medical and Chirurgical Society on February 25th, 1890,[1] brought forward strong evidence for believing that when a total transverse lesion of the spinal cord above the lumbar enlargement occurred, the results were complete paraplegia of motion and sensation, flaccidity of the lower extremities, with loss of tendon phenomena (knee-jerks, ankle-clonus), even though the lumbar enlargement remained healthy. His views were supported in a paper read on a subsequent occasion before the same Society by Mr. Bowlby[2] from observations made in several cases of fracture-dislocation of the spine. I was present at the meeting of the Royal Medical and Chirurgical Society at which Dr. Bastian's paper was read; and as the patient, E. D., was in Guy's Hospital at the time, I went carefully into his condition. I was rather surprised to find that, while the loss of motion and sensation in the lower extremities was complete, and though the knee-jerks were absent, yet there

[1] *Vide* 'Transactions,' vol. lxxiii.

[2] *Vide* same vol. 'Trans.'

was more or less rigidity. The explanation I proposed to myself was that there was a total lesion in the dorsal region, but that before the lesion had become total the muscles of the lower extremities had got too shortened to subsequently admit of full extension. But the microscopical examination of the compressed portion of the cord showed that the lesion was by no means total; nor was the lumbar enlargement affected, except as regards the descending degeneration in the crossed pyramidal tracts. The pigment found in the cells of the anterior cornua in the lumbar enlargement was also to be observed in those of the cervical enlargement; yet the patient's upper extremities were never more affected than could be accounted for by his chronic wasting disease. The presence of such pigment in nerve-cells is associated with old age.[1]

The first explanation of the absence of the knee-jerk that would occur to any one reading the account of this case, would be that the obtaining of the jerks was prevented by the contraction of the hamstring muscles. I was perfectly well aware of this objection at the time, but was certain that, until within the last few weeks of the patient's life, the hamstrings were never sufficiently contracted to hinder the knee-jerk. Moreover, there was no attempt at a knee-jerk, not even a slight contraction of the quadriceps extensor muscle, when its tendon was struck. I have recently inquired into the question of the hindrance of jerks by contraction of opposing muscles, by examining several cases of chronic hemiplegia and paraplegia with rigidity, and I find that the vigour of the knee- or elbow-jerk varies with the amount of rigidity; and that even in cases where the rigidity is excessive the quadriceps extensor (for example) can be seen to contract when its tendon is struck, though the opposing contraction of the hamstring muscles prevents the leg from being jerked out in the usual way.

In addition to the absence of the knee-jerks in this case, it is to be noticed that ankle-clonus could never be elicited. In short, there were none of those exalted tendon-phenomena that usually accompany spastic paraplegia. This particular

[1] Obersteiner, 'The Anatomy of the Central Nervous Organs,' &c., Eng. trans., p. 123.

case neither supports nor is antagonistic to Dr. Bastian's views; for though there were loss of knee-jerks and complete paraplegia of motion and sensation, the lesion in the cord not being total, yet there was gradually increasing rigidity.

Other points of interest to be noted with regard to this case are (1) that complete loss of motion and sensation in paraplegia does not necessarily mean total destruction of the cord; and (2) that descending degeneration in the pyramidal tracts does not of necessity give rise to increased tendon-phenomena, quite apart from the question of total transverse lesions of the cord. Why in this patient there was so marked an absence of knee-jerks and ankle-clonus I am unable to say.

TWO DOUBTFUL CASES OF TUBERCULOSIS,

IN WHICH THE DIAGNOSIS WAS CLEARED UP BY INOCULATION.

By J. W. WASHBOURN, M.D.,

AND

W. H. A. JACOBSON, M.Ch.

Case 1. *Cystitis.*—M. A. P—, æt. 32, was sent to Mr. Jacobson by Dr. Forty of Wootton-under-Edge, Gloucestershire on December 23rd, 1889, for obstinate cystitis. One brother is "consumptive," and mother "suffers from her chest." Father "asthmatic."

In June, 1888, patient first noticed pain at the end of micturition, and also (but to a less degree) when the bladder was filling. In December, 1888, the pain had become so severe that the patient could not hold her water at all, having to pass it every five minutes. This went on, with slight relief from treatment, till May, 1889, when patient had an attack of pleurisy in the right side. When she had recovered from this she was admitted into the Bristol General Hospital, where the bladder was washed out daily. In August, 1889, she had pleurisy in the left side. The bladder trouble had continued, small clots had been occasionally seen, but bleeding had not been a prominent feature.

On admission.—Patient now passes water about once an hour, occasionally six times an hour. The pain is worse as

the bladder fills, and is increased by getting about. There have been two or three slight attacks of retention. The urine is slightly acid, sp. gr. 1020, and contains a large flocculent deposit, proved to be pus. Neither blood, nor cells indicative of malignant growth, nor tubercle bacilli could be found.

January 7th, 1890.—The urethra being rapidly dilated the bladder was examined with the finger. It was small and contracted, but, save that it felt distinctly "pulpy," and its mucous membrane thickened and soft, the examination was entirely negative. The bladder was painted with a solution of silver nitrate (ʒij—ʒj).

16th.—As a result of the above treatment the patient can now hold her water much better both by day and night. She now only passes it three or four times in the daytime and once at night, as compared with four or six times every night, and about every hour in the day.

The application was repeated on January 24th, and patient went out on February 6th. She had lost all pain both during micturition and when the bladder was full. She now only passes water four times during the day, and about three times in the night. There had been no blood by microscopical examination since January 13th, but up to January 31st (the last note on this matter) pus was present in the field.

31st.—A specimen of urine was sent to the Bacteriological Laboratory to be examined. Dr. Washbourn failed to find any tubercle bacilli in the sediment by microscopical examination. A small portion of the sediment was accordingly placed under the skin of the back of a guinea-pig. In three weeks' time a gland in the left groin was observed to be somewhat enlarged. With the exception of a slight increase in size of this gland, the animal continued perfectly healthy.

On May 9th it was killed by means of chloroform.

Post-mortem the body was found to be well nourished and the organs healthy. There was a gland in the left groin, enlarged to the size of a nut and full of caseating material. Cover-glass preparations made from the caseating material revealed the presence of tubercle bacilli.

Case 2. *Vesical calculus; lateral lithotomy; subsequent caseation of wound and inguinal glands; scraping out of these; recovery.*—D. M—, æt. 10, was admitted under Mr. Jacobson with the usual symptoms of stone in the bladder, August 16th, 1890. He had lived at Aberystwith all his life. Incontinence of urine, slight hæmaturia, and pain in the penis had been more or less marked for seven years.

On admission pain is the chief feature, the child lying doubled up in bed, constantly crying. A sound readily detected a rough and relatively heavy stone. Lateral lithotomy was performed with a straight staff on August 17th, and a vesical calculus (lithic acid and lithates), weighing 300 grains, removed. Mr. Jacobson did not see the child between August 29th and October 2nd. The report states that urine was first passed by the penis on September 2nd, and that it all came naturally on September 13th.

On the 23rd a little urine again came through the wound, and the inguinal glands on both sides were found to be enlarged.

When Mr. Jacobson saw the patient on October 4th, the inguinal glands on the right side were enlarged, tender, and matted together; those on the left side were in a similar but advanced condition, suppuration having evidently taken place.

On October 10th, A.C.E. mixture being given, the lithotomy wound was scraped out thoroughly with a sharp spoon. Much ill-favoured, flabby granulation tissue was thus removed with numerous caseating patches. A few days later the suppurating glands in the left inguinal region were scraped out with a like result.

The wound healed rapidly, and the child went out November 14th, with a superficial sinus in the perinæum and the left groin.

October 10th, 1890.—A small portion of the scraping was sent to the Bacteriological Laboratory for examination. Dr. Washbourn found that it consisted of granulation tissue with minute caseating foci. No tubercle bacilli could be found. A small portion of the tissue was placed into the anterior chamber of the eye of a rabbit under chloroform.

On November 1st a minute reddish-grey tubercle was

seen at the seat of inoculation. Two very minute greyish masses were seen in the iris.

On the 18th the tubercle at seat of inoculation was the size of a large shot, and several minute grey tubercles were scattered over the iris.

On January 27th, 1891, the animal was killed by means of chloroform.

Post-mortem the body was found well nourished, and the organs healthy. There was a large tubercle in the eye at the seat of inoculation, and several minute ones scattered through the iris. Sections showed the presence of tubercle bacilli.

These two cases show the importance of inoculating animals in order to prove the nature of doubtful cases of tuberculosis. If tubercle bacilli are present in moderate number, their presence can be detected by microscopical examination, and a diagnosis be made at once with certainty. If, on the other hand, the bacilli are scanty, even a prolonged microscopical examination may fail to reveal their presence, as was the fact in the above cases. A diagnosis can then only be arrived at by cultivating the bacilli, either within the living body of susceptible animals or on suitable media, such as blood-serum or glycerine agar. The latter method presents, as is well known, so many difficulties that a negative result is valueless. The former method therefore should be adopted, and the inoculation may be performed in two ways, the suspected material being introduced either into the anterior chamber of the eye, as in Case 2, or in a little pocket under the skin, as in Case 1. If the material is contaminated with pyogenic or septic organisms, as is the case with sputum or pus, it should be introduced under the skin ; otherwise it is much preferable to inoculate into the anterior chamber of the eye, where the formation of tubercles in the iris can be so easily seen. As soon as any distinct local manifestations of tubercle appear (3—4 weeks) the animal may be killed and the affected tissue examined microscopically. The presence of tubercle bacilli in these tissues proves the tuberculous nature of the material inoculated.

After-history of the above Cases.

Case 1.—Pain and frequency in micturition gradually returned, and the patient again came under my care at the Royal Hospital for Children and Women in April, 1890. On examining the bladder with the cystoscope the mucous membrane was everywhere swollen, pulpy-looking, and its colour either a deep red or a livid purple, nowhere of the natural pinkish hue. Numerous short tags of necrotic mucous membrane were seen floating up off the region of the trigone, and adherent to it, but the fluid was rendered so persistently murky by the pus which constantly streamed up and diffused itself through each "washing" used, that I was unable to satisfy myself whether the above tags pointed, as I suspected, to the edges of tubercular ulcers. This, it will have been seen, was cleared up a little later by the result of Dr. Washbourn's experiment. Silver nitrate (ʒij to ʒj) was again applied, and the patient went out in May with painless micturition and normal control over her bladder. As soon as Dr. Washbourn's inoculation had made the nature of the case plain, I wrote advising her to return as soon as her micturition again became frequent and painful, that a suprapubic cystotomy might be performed, and any ulceration treated by scraping and iodoform, as used so successfully by Mr. Battle in his case of "Tubercular Ulceration of the Bladder" ('Clin. Soc. Trans.,' vol. xxiii, p. 201). Dr. Forty kindly wrote to me, in March, 1891, to say that the patient's general health was excellent, and that she had made flesh. As, however, the frequency of micturition has latterly returned, the outlook is a very grave one.

Case 2.—Wishing to know the result here, I wrote to Dr. J. D. Harris, of Aberystwith, who kindly saw the boy, and wrote (February, 1891) that the sinuses resulting from the scraping out of the lithotomy wound and the left inguinal glands had healed firmly very soon after the return of the patient to the air of Aberystwith, in November, 1890, and that the child had remained quite well in every respect since.

How the lithotomy wound became infected remains a mystery. The child had no other evidence of tubercular trouble about him, and no evidence in this direction was obtained from the family history.

ABNORMALITIES

OBSERVED IN THE

DISSECTING ROOM OF GUY'S HOSPITAL

DURING THE

SESSIONS 1888—1890.

By L. A. DUNN, M.S., J. W. WASHBOURN, M.D.,

AND

J. H. TARGETT, M.S.

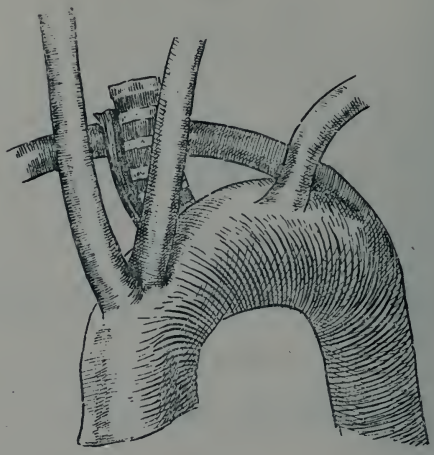
I. Arteries.

Head and Neck.

Absence of the innominate artery.—In one subject an absence of the innominate artery was noted. The right and left common carotids in this case arose close together from the arch of the aorta on the right of the trachea, consequently, in order to reach its normal position, the left common carotid artery crossed over in front of the trachea. Both subclavian arteries arose from the aortic arch near together, to the left of the trachea, the right subclavian being more to the left. This then passed across the front of the cervical spine behind the trachea and œsophagus to supply the right upper extremity (Fig. 1).

Thyroid axis.—In one subject the posterior scapular artery,

Fig. 1.



on both sides, came off from the third part of the subclavian artery. It perforated the scalenus medius, passed between the cords of the brachial plexus, and gave origin to the supra-scapular artery.

In two instances the transversalis colli artery arose from the ascending cervical branch of the inferior thyroid.

In another subject a large branch was given off, from an otherwise normal supra-scapular artery, at the outer margin of the scalenus anticus muscle. It formed a loop downwards, and then passed upwards and forwards superficially across the first part of the subclavian artery, the internal jugular vein, and common carotid artery, but deeply to the sterno-mastoid, sterno-hyoid, and sterno-thyroid muscles. It reached the front of the trachea, and ended in the superficial fat.

We also observed a large branch which sprang from the right inferior thyroid artery. It passed downwards behind the carotid sheath, till it reached the innominate artery, and then continued its course deeply between the innominate artery and trachea for about half an inch; it then suddenly turned upwards upon the front of the trachea, and supplied the inferior aspect of the thyroid isthmus.

Superior thyroid artery.—In two cases this artery arose from the common carotid half an inch below its bifurcation.

In one of these the only branch besides the terminal was the superior laryngeal, which reached the interior of the larynx by perforating the thyroid cartilage; in the other the superior laryngeal was given off by the external carotid artery.

Facial artery.—This was a branch of the temporal artery, apparently an enlarged transverse facial. It passed forwards, having a very tortuous course till it reached the angle of the mouth, when it assumed the position of the normal facial artery. The facial vein in this subject was quite regular.

Meningeal arteries.—In one subject the middle meningeal artery on each side arose from the ophthalmic. It passed backwards through the sphenoidal fissure, and then outwards along the under surface of the small wing of the sphenoid, to assume its usual position on the parietal bone.

In another case the middle meningeal artery passed through the foramen ovale; whilst in a third there was a large posterior meningeal branch of the occipital artery, quite as large as the ordinary middle meningeal, which entered the skull by means of a large mastoid foramen.

Upper Extremity.

Posterior circumflex artery.—In two instances the posterior circumflex artery arose from the brachial in common with the superior profunda. It passed backwards below, and then upwards behind, the tendon of the teres major muscle to reach its usual position. In a third, the anterior and posterior circumflex arteries arose with the superior profunda artery by a common trunk from the axillary artery opposite the lower border of the subscapularis tendon.

High division of the brachial artery.—This took place opposite the insertion of the coraco-brachialis muscle. The external branch passed downwards in the position of the normal brachial artery, but at the bend of the elbow it divided into the radial recurrent, the common interosseous, which gave off the anterior and posterior ulnar recurrent arteries, and a fine trunk in the position of the normal radial artery. The inner trunk passed downwards and inwards in front of the internal condyle of the humerus and the attached muscles, and

divided, about one inch below the condyle, into ulnar and radial arteries, both of which ran superficially, the only muscle in front of them being the palmaris longus, which crossed the ulnar artery about the middle of the forearm.

Comes nervi mediani.—This artery, which was unduly large, arose from the radial opposite the lower border of the pronator radii teres muscle.

Abdomen.

Abnormalities of the cœliac axis and its branches.—In one subject we noticed an absence of the cœliac axis. The splenic artery in this case arose from the abdominal aorta, and gave off the gastric branch and one to the liver. The hepatic artery arose from the superior mesenteric.

In another subject the hepatic artery arose normally from the cœliac axis, but only supplied the left lobe of the liver. The larger branch arose from the superior mesenteric, and supplied the right lobe of the liver and the gall-bladder.

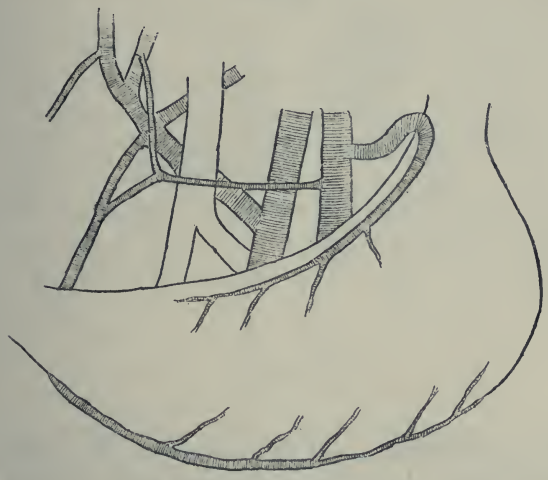
In a third we noticed the following distribution of the branches of the cœliac axis :—One passed to the left like the normal splenic artery ; a second gave upwards one branch to the left lobe of the liver, which entered the transverse fissure, and several smaller branches, which passed along the smaller curvature of the stomach ; whilst a third passed to the right, behind the first portion of the duodenum, and divided into the superior pancreatico-duodenal, pyloric, and right gastro-epiploic arteries ; the right lobe of the liver being supplied by a branch of the superior mesenteric, which passed upwards in front of the foramen of Winslow like the normal hepatic artery.

In a fourth the main trunk of the cœliac axis divided into splenic and gastro-duodenal branches ; the latter gave off three trunks, the superior pancreatico-duodenal, pyloric, and right gastro-epiploic. The gastric arose directly from the aorta, about a quarter of an inch to the right of the cœliac axis, and the hepatic came from the superior mesenteric artery.

In a fifth the arrangement of the vessels is indicated in Fig. 2. The hepatic artery arises from the superior mesen-

teric, and passes behind the portal vein (unshaded). The gastric artery arises separately from the aorta. A small artery from the aorta crosses in front of the portal vein, and divides to join the gastro-duodenal and right branch of the hepatic artery.

Fig. 2.



We also observed both inferior phrenic arteries arising from the cœliac axis.

In one instance the splenic artery gave origin to a large branch, which passed from left to right between the layers of the great omentum, parallel with, but three inches below, the left gastro-epiploic artery (the two vessels being joined by numerous fine connecting branches), and ended by joining the right gastro-epiploic.

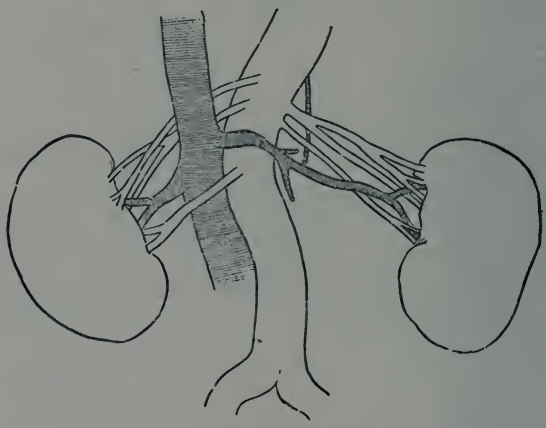
Renal arteries.—Abnormalities were noticed in three subjects. In the first the right kidney was supplied by three arterial trunks arising separately from the aorta ; the upper two passed behind the inferior vena cava, whilst the lowest and largest, passed in front of that vessel. The left kidney was supplied by two separate trunks arising from the aorta, the upper of which bifurcated about half an inch from its origin (*vide* Fig. 3. The veins in this drawing are shaded).

In the second the right kidney had two arteries, the upper being the normal renal, the lower arising from the aorta just

above its bifurcation, and supplying the inferior extremity of the organ.

In the third the left kidney was supplied by two large arterial trunks which arose from the aorta.

Fig. 3.



Sacra media artery.—In four subjects the sacra media artery arose from the aorta in common with the last lumbar artery on the left side.

Obturator artery.—In four instances this artery had an abnormal origin. In two it arose from the deep epigastric artery, in one it was a branch of the external iliac artery, and in one it came off from the posterior division of the internal iliac artery. In the first two cases the artery passed internal to the crural ring.

We also noticed an accessory internal pudic artery coming off from an otherwise normal obturator artery.

Lower Extremity.

Accessory circumflex iliac artery.—This arose from the outer side of the external iliac artery, equal in size to, and about one inch above, the deep circumflex iliac artery. It passed outwards over the iliacus muscle supplying it, and ended by anastomosing with the deep circumflex iliac artery at the iliac crest.

Profunda femoris artery.—This arose from the inner

aspect of the common femoral artery, half an inch below Poupart's ligament. It passed inwards in front of the common femoral vein, and, having arrived at the inner side, went behind the vein, and then assumed its normal course.

External circumflex artery.—In two cases the external circumflex artery arose from the superficial femoral artery, and crossed in front of the profunda artery in its course outwards.

Internal circumflex artery.—The internal circumflex artery arose from the deep epigastric artery on the left side, and passed downwards internal to, and behind, the common femoral for about one inch, to reach the interval between the psoas and pectineus muscles.

In another subject the internal circumflex artery sprang from the common femoral, which divided into three branches, viz. the superficial, and deep femoral, and external circumflex arteries.

Veins.

Inferior vena cava.—This vessel was placed on the left side of the abdominal aorta as high as the second lumbar vertebra, when it crossed to the right in front of the artery. The junction of the tributaries on the left was precisely similar to the usual arrangement on the right side.

Renal vein.—The left renal vein in one subject passed to the right beneath the abdominal aorta, and then gave origin to a trunk, equal in size to itself, which passed up into the thorax through the aortic opening in the diaphragm, and became continuous with the great azygos vein.

Muscles.

Head and Neck.

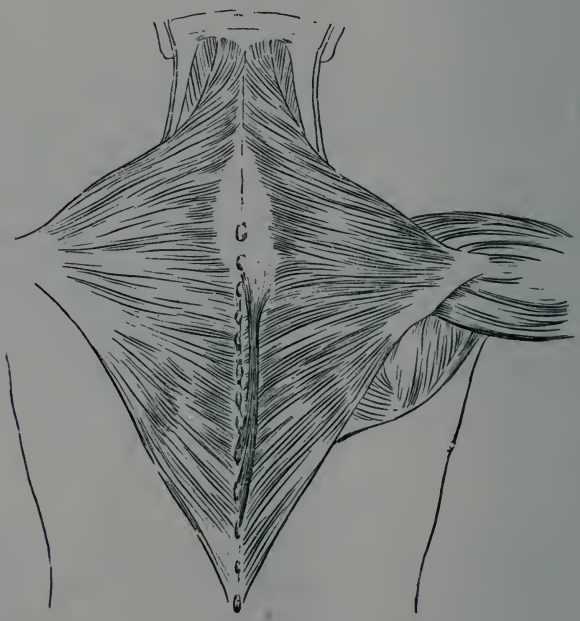
The trapezius.—In two instances connecting fibres between the trapezius and sterno-mastoid were observed. In one there was a flat fleshy fasciculus, on the right side of a male subject, running from the lower end of the anterior border of the trapezius to the front of the sternal third of the clavicle. It crossed over the subclavian triangle, having

the cervical fascia and the descending cervical nerves beneath it, and ended in a rounded tendon in front of the outer border of the clavicular head of the sterno-mastoid.

In the second instance, also on the right side of a male subject, the muscular slip arose in a similar manner from the trapezius, but was inserted into the upper margin of the clavicle *behind* the clavicular origin of the sterno-mastoid. The external jugular vein turned round its lower margin close to its attachment to the trapezius.

The usual origin of the trapezius from the superior curved line of the occipital bone was wanting in one subject, the muscle ending in a pointed extremity at the external occipital protuberance. But in this case the insertion of the sterno-mastoid into the superior curved line was unusually wide.

Fig. 4.



Mr. Davies-Colley contributes the following abnormality from a dissection made for his lectures on muscles (Fig. 4).

Trapezius.—In a body used for the lecture theatre, a band

of muscle was found on the right side, about half an inch broad, running vertically upwards near the spine, and upon the trapezius. Its origin was by short tendinous fibres, about a quarter inch long, attached to the spines of the eighth, ninth, and tenth dorsal vertebræ. After a course of about four inches, in which the band lay with its inner border parallel to, and half an inch from, the spines of the vertebræ, it became tendinous, and was inserted into the fascia covering the trapezius, and into the lower part of the oval aponeurosis of that muscle, close to and on a level with the spines of the second and third dorsal vertebræ.[1]

The digastric—In two subjects this muscle had an additional anterior fleshy belly, arising from the median raphé between the lower jaw and the hyoid. In one of them it was present on both sides. In shape it was triangular, with the base towards the middle line, and situated immediately below the symphysis of the jaw.

The following abnormality is probably of rare occurrence: *Muscular slip between tendon of digastric and cartilago*

Fig. 5.

[1] I am informed by my friend Professor Thane that this slip is described as a

triticea.—On the right side there was a muscular slip, an inch and a quarter long and a quarter of an inch broad, arising from the *cartilago triticea* and the posterior thyro-hyoid ligament (which was very cartilaginous), and extending upwards and forwards over the great cornu of the hyoid and the posterior inferior angle of the hyoglossus, to the lower margin of the tendon of the digastric. The hypoglossal nerve was lying on this muscular slip. The flat tendon of insertion of the stylo-hyoid passed entirely behind the tendon of the digastric to be attached to the small cornu of the hyoid. The anterior belly of the omo-hyoid on the same side had an additional attachment to the upper end of the oblique line on the thyroid cartilage (Fig. 5).

Wide origin of omo-hyoid.—On the left side of a muscular subject the omo-hyoid arose as a broad muscular sheet from the posterior surface of the clavicle, the inner margin of the conoid ligament and a fibrous arch stretching from this point to the supra-scapular (transverse) ligament. Its origin from the scapula was normal. The subclavius was normal.

This muscular sheet is normally represented by a fibrous expansion, which is "a portion of the lower border of the omo-hyoid expanded and blended with the overlying portion of the cervical fascia" (Macalister).

In another subject there was a duplicate omo-hyoid on the right side. It was lying below the normal muscle, and it arose from the hyoid in conjunction with the sterno-hyoid, while at its insertion into the scapula it was placed just external to the attachment of the normal muscle.

As the sterno-hyoid is so closely related to the omo-hyoid, it may be noted here that complete absence of the *former* muscle was observed in one instance. There was an unusually broad anterior belly of the omo-hyoid, but no vestige of a sterno-hyoid. The sterno-thyroid was normal, as well as all the muscles on the opposite (left) side.

rudiment of the panniculus carnosus by Sir W. Turner and Mr. J. B. Perrin, in vol. v of the 'Journal of Anatomy,' pp. 116 and 241.—N. D.-C.

Upper Extremity.

Musculus presternalis (Testut).—Two examples of this muscle were met with. One specimen consisted of a small muscular slip, two and a half inches long, which arose from the aponeurosis of the external oblique over the upper end of the rectus abdominis on the sight side. It passed upwards and inwards to end in the fascia over the front of the first piece of the sternum. This slip was tendinous at either extremity.

In another subject this muscle consisted of a narrow band, half an inch broad, arising by slips from the cartilages of the second to the sixth ribs inclusive on the right side, close to the margin of the sternum. The lowest fibres were connected with the aponeurosis of the external oblique. The muscle passed upwards, parallel with the right border of the sternum, and terminated by crossing the first piece of the sternum obliquely, to blend with the highest slip of the sternal origin of the *left* pectoralis major. It was not present on the opposite side. The anterior cutaneous branches of the intercostal nerves were lying on it.

This muscle is rarely met with. It is stated to have occurred only thirty-three times in 920 subjects. That it is not due to a prolongation upwards of the rectus abdominis, is shown by the fact that such an abnormality invariably lies beneath the pectoralis major, while the presternalis is superficial to the sheath of the rectus, as seen in the above examples. Most authorities are now agreed that it represents certain displaced fibres of the pectoralis major, chiefly because it is supplied by the same group of nerves as the latter muscle. In seventeen out of twenty-two examples the nerve of supply was traced to one or other or both of the anterior thoracic nerves. Indeed, the musculus sternalis has been ably used by Prof. Cunningham as an illustration of the value of nerve-supply in determining muscular anomalies.

Incomplete forms of an occipito-scapularis (Wood).—On both sides of the neck a muscular slip was noted passing from the transverse process of the third cervical vertebra to the first serration of the serratus magnus. At its upper attachment it was joined to the normal origin of the levator

anguli scapulæ from that vertebra, and below it blended with the serratus magnus about one inch from the costal end of the first serration. These slips were of much the same size on the two sides. A similar fleshy slip, half an inch broad, was found in a male subject, connecting the upper margin of the serratus posticus superior with the lower margin of the levator anguli scapulæ on the left side.

Wood described the typical occipito-scapularis as a flat muscle situated beneath the trapezius. Its upper end was connected with the middle third of the superior curved line of the occipital bone, between the attachments of the trapezius and complexus, and internal to the splenius capitis. Below it blended to a variable degree with the scapular attachments of the rhomboid. Various incomplete forms of this muscle were enumerated by Wood, under some of which the two now recorded may be placed.

Coraco-brachialis brevis (Cruveilhier).—In a muscular female subject there was a bulky slip of muscle, one inch in breadth, passing from the prominent root of the right coracoid process to the upper end of the inner bicipital ridge. It was situated above about half an inch internal to the normal coraco-brachialis, and its lower end was lying beneath the tendons of the teres major and latissimus dorsi. In its course it passed at right angles over the upper end of the subscapularis, and was in relation with the circumflex arteries, so that the anterior and posterior branches were respectively above and beneath the muscle. From the anterior surface of this abnormal slip a thin bundle of fibres passed forwards over the cords of the brachial plexus to be attached to the costo-coracoid membrane. There was a precisely similar, though less developed, muscle on the left side, except that it wanted the slip to the costo-coracoid membrane.

Achselbogen muscle.—An incomplete variety of this muscle was observed on the left side of a well-developed subject. It consisted of a fleshy slip, three quarters of an inch broad, passing from the axillary border of the latissimus dorsi, two and a half inches from its insertion into the humerus, over all the axillary vessels and nerves, to join the combined tendon of the coraco-brachialis and biceps two inches

from the tip of the coracoid process. In the complete form it joins the tendon of the pectoralis major. It has been suggested by Calori that this muscle would favour the venous circulation in the limb by tightening the axillary and brachial fasciæ and compressing the axillary vein.

Biceps brachii with three heads.—Three examples of this condition were noted. In all the muscular slip arose from the internal surface of the humerus between the insertion of the coraco-brachialis and the attachment of the brachialis anticus. Most of the fibres ended below on the posterior surface of the tendon of the biceps, and the remainder were inserted into the inner border of the bicipital fascia.

On the right side of another subject, this muscle had an abnormal insertion. A small muscular fasciculus arose from its inner margin, about three inches below the shoulder-joint, and becoming tendinous, blended with the internal intermuscular septum, in which it could be traced as far as the supracondylar ridge.

Inversion of the palmaris longus was observed on one occasion, the lower half of the muscle being entirely muscular, while its upper portion consisted of a long thin tendon.

Duplication of this muscle was also met with, the additional fasciculus arising on the ulnar side of the normal muscle, and, passing beneath it, to be inserted into the fascia over the abductor pollicis, and into the anterior annular ligament. The two muscles were nearly equal in size and shape.

Additional origin of the first lumbricalis of the hand.—On the right side the first lumbricalis had its normal origin from the radial side of the flexor profundus digitorum tendon, and an additional muscular origin from the middle portion of the flexor longus pollicis in the forearm. This slip was directly continuous with that portion of the flexor longus pollicis which arises from the coronoid process of the ulna. It was muscular at its origin, and also where it blended with the first lumbricalis ; but there was an intervening portion of tendon where it passed over the wrist-joint.

As the lumbricales are considered to be displaced heads of the flexor profundus digitorum, this muscular slip may be looked upon as an incomplete displacement which has become

attached to the flexor longus pollicis. Its direct continuity with the additional head of the flexor longus pollicis from the coronoid process of the ulna is interesting.

On the opposite side the first lumbricalis arose entirely from the tendon of the flexor longus pollicis.

Supplementary abductor minimi digiti.—In the left forearm of a male subject there was a large muscular belly, three and a half inches in length, situated on the outer side of the flexor carpi ulnaris, and arising by a tendinous expansion from the deep fascia of the forearm covering this muscle and the adjacent palmaris longus. It passed downwards along the outer side of the pisiform bone to be inserted by a rounded tendon into the inner side of the base of the first phalanx of the little finger, in company with the abductor minimi digiti. The fibres of the latter muscle were, in fact, partly inserted into this rounded tendon. The palmaris brevis was superficial to it as it crossed the hypothenar eminence.

Extensor mediani et annularis manus.—On the left side of a very muscular subject there were two small muscles

Fig. 6.

arising from the fascia over the back of the carpus, and inserted into the third and fourth digits. That to the third digit crossed the third metacarpal bone and joined the expansion of the dorsal interossei on the radial side ; while that to the fourth digit was much smaller in size, and was inserted on the ulnar side (Fig. 6).

Lower Extremity.

Absence of quadratus femoris and inferior gemellus.—These muscles were entirely absent on the left side of a female subject, so that the obturator externus and the terminal branches of the internal circumflex artery were lying exposed beneath the gluteus maximus. On the same side an abnormal muscle was found within the pelvis, arising just below the brim and in front of the attachments of the sacro-iliac ligaments; it had also a slight connection with the fascia covering the outer part of the sacral plexus. This muscle passed through the great sacro-sciatic foramen, in front of the pyriformis, and superficial to the extreme posterior fibres of the gluteus minimus ; from both of these it was separated by cellular tissue. Beyond the foramen it was situated *above* the great sciatic nerve, and running outwards in company with the obturator internus and superior gemellus, it was inserted with them into the great trochanter by a distinct rounded tendon. At the back of the hip-joint there was a large articular nerve proceeding from the sacral plexus, and lying in the exact position of the nerve to the quadratus femoris. Normally the articular branch from this nerve is but a fine filament. The additional fasciculus described above was probably a portion of the pyriformis sheet of muscle, which had become separated by the passage through it of the great sciatic nerve.

Third head to gastrocnemius.—In a negro, aged 26, there was an additional head to the right gastrocnemius arising in two portions, the internal of which was a rounded fleshy mass attached to the centre of the popliteal space of the femur, and the other a thin muscular slip from the external condyle of the femur just internal to the well-developed

belly of the plantaris. These two portions united to form a thick mass, which blended with the inner head of the gastrocnemius at the angle of junction with the outer head. The popliteal artery was situated on the inner side of this additional muscular slip (Fig. 7).

Fig. 7.

On the left side there was a very similar muscle, also on the outer side of the popliteal artery. In this instance there was a fleshy portion from the centre of the popliteal space, and a second internal slip arising near the inner head of the gastrocnemius beneath the artery. These two portions blended with the outer head of the gastrocnemius, but not at the angle. The left plantaris was also well developed.

Additional origin of flexor accessorius.—This was a long, thin, fleshy slip arising from the fascia over the tibialis posticus, two inches above the ankle-joint, and passing down-

wards to the tendon of the flexor accessorius. It was lying on the tendon of the flexor longus hallucis, though separated from it by a distinct sheath, and it joined the tendon of the accessorius in company with the process from the flexor longus hallucis.

Flexor longus digitorum pedis.—In two other subjects this muscle had an additional origin, similar to that described above; that is, from the tough fascia covering the tibialis posticus. The fleshy slip was two inches broad, and arose about three inches above the level of the ankle-joint; and arching over the tendon of the tibialis posticus, it joined the flexor longus digitorum behind the internal malleolus. This anomaly, common to the two muscles, is of interest as an indication of their intimate relationship.

A CASE

OF

INTERNAL DERANGEMENT OF THE KNEE-JOINT,

FROM SPLITTING OF THE INTERNAL SEMILUNAR CARTILAGE, TREATED BY OPERATION.

By N. DAVIES-COLLEY.

The following case seems to me worthy of record, as an example of a rare form of injury, and of a somewhat novel mode of treatment. In the recorded examples of internal derangement of the knee-joint, the usual lesion has been separation of one or other semilunar cartilage from its attachment to the tuberosities of the tibia and the lateral ligaments. Successful results have been obtained in some cases by suturing the cartilage to the periosteum of the adjacent bone, in others by removal of considerable portions of the loosened cartilage. I am not aware that any one has hitherto had to treat a lesion consisting of splitting up of the cartilage itself. At the time of the operation I was in much doubt whether so evascular a structure as the semilunar cartilage would show any capability of repair; and I was, therefore, very doubtful whether it would not be wiser to remove the loose strips altogether. Judging from the successful results which have followed removal of portions of the

fibro-cartilage in other cases,[1] I am disposed to think now that this would have been the safer plan, and if I had to operate again in such a case I would adopt it. The method which I adopted in this case of stitching the strips to one another and to the still attached portions of the cartilage by means of chromicised catgut proved successful; but the patient ran some risk through the acute synovitis which was set up in the joint. There was never any general suppuration, but for more than a week large masses of coagulated fibrin were discharged from the interior of the articulation, and there was a good deal of fever.

The subsequent progress of the case was very satisfactory. The inflammation all subsided, and left the joint unimpaired. For a time the patient, as a precaution, wore a support, but he was soon free from pain and stiffness; and a year and a half after the operation, he wrote to say that he was able to use the joint in rather violent exercise.

Mr. George K. D—, æt. 21, a young gentleman who had been bush-farming in Australia, was brought to me in October, 1887, by my friend Mr. Hugill, suffering from pain in his left knee-joint and lameness.

Six years before, the horse he was riding reared and fell back upon him, crushing his left knee. Considerable swelling of the joint followed, and the limb was put up in splints. In two months he was all right again, though he thinks that his knee was somewhat weaker. Six months after the injury he was playing lawn-tennis, and, as he turned to make a back-hand stroke, he felt the knee suddenly give way. It appears to have been dislocated outwards in a somewhat flexed position. He had to lie down until the doctor came, who reduced the deformity without an anæsthetic, but after much painful manipulation of the limb. Since that time the joint has always been weak, and has given him a great deal of trouble. He has had several bad attacks of synovitis, but these of late have not been quite so severe. From time to time the knee gets suddenly locked, being on these occasions somewhat flexed and displaced outwards, as it was after

† *Vide* case of Mr. Croft and others cited by him in 'Clinical Society's Transactions,' vol. xxi, p. 159.

the lawn-tennis accident. Usually, by taking hold of his leg below the knee, and by first extending the joint completely and then flexing it, the displacement disappears. He says that when it is out a projection or a transverse ridge is visible along the upper border of the inner tuberosity of the tibia. His medical attendant thought that this swelling was due to a projection of the displaced semilunar cartilage, but from questioning the patient it would appear to be merely the upper edge of the bone thrown into greater prominence by the retirement of the cartilage into the interior of the joint. The last time he locked his knee was last night, when in suddenly turning to the right his foot caught for a moment upon a thick carpet. The pain caused by this displacement is often very great. In Australia he had to ride a great deal, and often upon very imperfectly broken horses. This displacement used frequently to occur while he was on the saddle, and the pain and weakness thus produced were often so great that he had to roll off the back of the horse and fall upon the ground.

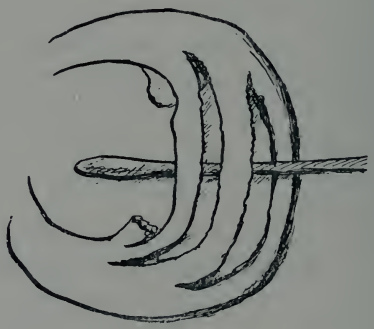
The patient is rather above the middle height, of spare and wiry frame, and, with the exception of his knee-trouble, in very good health. There is nothing abnormal to be seen or felt about the joint, which is well formed and not at all swollen. There is tenderness on pressure upon the middle of the upper edge of the inner tuberosity of the tibia. He has tried knee-caps, but they do not prevent the displacement, and make it far more difficult to reduce when it does occur.

I suggested that fixation of the limb in the extended position for five or six months might be of benefit, but on the whole I thought that it would be wiser to resort at once to an exploratory operation, as I had very little doubt that some injury to, or partial separation of, the internal semilunar cartilage was the cause of his symptoms. He preferred the latter course, and was admitted into one of the cubicles of Bright Ward under my care on October 20th, 1887.

On October 22nd, a carbolic compress having been applied to the knee for a few hours, ether, preceded by a little chloroform, was administered. Under careful antiseptic precautions, such as the use of the spray and shaving of the limb and washing it with perchloride of mercury solution

(1 in 1000), I made a transverse incision three inches long from the inner border of the ligamentum patellæ along the inter-articular plane nearly to the middle of the inner side of the joint. There was but little hæmorrhage. Some vessels had to be twisted, and others tied with chromicised gut.

Upon opening the joint at first nothing abnormal could be seen, but on bending the knee through about 60° I exposed to view the internal semilunar cartilage, which presented the appearance which I have indicated in the figure by a rough sketch made from memory immediately after the



operation. The front part of this cartilage was of normal appearance, but at the point where it turns to go backwards it was completely split by a fissure running parallel to its free border, and an inch and a quarter long. The width of the strip thus separated, except at its ends, from the rest of the cartilage, was about an eighth of an inch. A quarter of an inch behind the commencement of the first fissure was another fissure also running parallel to the margins of the cartilage, but an eighth of an inch nearer to the attached margin than the previous one. It extended backwards to the same level as the other, and was about one inch long. An interval of about a tenth of an inch separated it from the attached border of the cartilage. There appeared to be also some horizontal splitting of the cartilage, for a small partly-separated piece of fibro-cartilage, about a sixth of an inch in its longest dimension and a twentieth of an inch thick, lay beneath

the anterior extremity of the first strip, and a similar semi-detached fragment of rather larger dimensions was just visible beneath the posterior extremities of the strips. I was, however, unable to examine accurately into this part of the injury, as it was in a great measure concealed in the deep recess at the back of the joint. The splitting of the cartilage was almost entirely in the antero-posterior direction, and appeared to terminate at the point where the cartilage changes from a backward to an outward direction. The point of a probe could easily be passed, as in my sketch, underneath the two strips which lay quite loosely upon the articular surface of the tibia.

After snipping off with the scissors the small projection which lay beneath the anterior extremities of the two strips, I then proceeded to stitch together the strips with chromicised gut (No. 1). I first attached that strip which lay nearest the free border to the middle one, and then the middle strip to that portion of the cartilage which lay close to its attached border. I now proceeded with the same kind of suture to stitch together the edges of the synovial membrane, and afterwards the edges of the divided aponeurosis of the quadriceps tendon. Lastly, I put seven or eight small sutures into the skin. No drainage-tube was inserted. The wound was now dressed with a thick covering of carbolic gauze, and a plaster-of-Paris splint was applied from the foot to the top of the thigh. The operation lasted rather more than one hour.

From the comparison of the condition found in the joint and the history of the case, I came to the conclusion that when the horse fell upon his knee six years before, it must have struck his leg low down upon the outer side, and forcibly adducted the knee in such a way as to compress the inner part of the internal semilunar cartilage between the condyle and the tuberosity, splitting it longitudinally. As a result of the two months' rest some sort of union probably took place between the edges of the lacerated cartilage. Six months later the sudden twist in his knee must have separated the adhesions which had formed. Since then these strips must have been frequently displaced, and by getting between the internal condyle and inner tuberosity

they must have caused the locking of the joint, followed by the attacks of synovitis, from which he had so frequently suffered.

October 23rd.—He has been in great pain all the night; but little relief was given to him by morphia injections, and he only had two or three hours' sleep. I slit up the plaster-of-Paris splint, which he said was too tight. The lower part of the leg was somewhat cold. There was no swelling of the joint. I changed the dressing, upon which was about half an ounce of bloody serum. The posterior tibial artery was pulsating freely, but I could not feel the dorsalis pedis. I was rather afraid that he was suffering from the effects of too tight bandaging.

24th.—Still very restless, but not in so much pain. Has been taking small quantities of Tr. opii. Pulse 120, morning temp. 100·4°; evening temp. 101°.

25th.—Much more comfortable. Second dressing. Some redness around the knee, which is a little swelled. About two ounces of bloody serum.

26th.—Quite comfortable. Discharge has come through. Third dressing. Two ounces of slightly turbid serum. Inner side of knee somewhat swollen, and upon pressure turbid serum flows from the anterior portion of the incision.

27th.—Did not sleep last night. Is somewhat restless. No discharge. Opened up the middle of the wound, divided three or four sutures, and let out an ounce of serum with a few flakes of lymph. Put a small drainage-tube into the superficial portion of the wound but not into the joint.

28th.—Complains of a dull aching cramp in his calf, and is rather restless. Temperature below 100°.

29th.—Not quite so well. Temperature has been over 101° in the night. About one ounce of bloody serum upon the dressings. Joint looks a little swollen. I passed in a probe, and about half an ounce of clear serum with a little blood came away.

31st.—Still has to be dressed daily, as a quantity of thin serous discharge comes through the dressings. A large coagulum of clear jelly-like substance covering the edges of the wound. At least three ounces of discharge upon the dressings. No smell.

November 1st.—He is very restless and sleeps badly. Last night while asleep he kicked his cradle on to the floor, and appears to have somewhat shaken his limb, as ever since he has had a good deal of pain in the calf and hip. About two ounces of discharge upon the gauze. The serum is somewhat turbid and the jelly-like coagulum more opaque. The joint has still a puffy appearance.

2nd.—Still the same peculiar discharge of fibrinous material. He complains of a good deal of pain in various parts of the limb. The plaster-of-Paris splint was removed, and a long back-splint applied with a foot-piece, and also with an interruption six or eight inches long opposite the knee, in order that a mixture of extract of belladonna and glycerine might be applied to the ham, which was painful.

4th.—The new splint is more comfortable, but he has to have injections of morphia to procure sleep. Temperature still rises to over 100° in the evening. The wound is healing. There is still a discharge of nearly an ounce of somewhat opaque jelly upon the dressings. The knee hardly, if at all, swollen. He was yesterday allowed to sit up for his bed to be made.

7th.—Dressed after two days. Not more than half a drachm of serous discharge. Joint is but little swollen. Skin over it is cool, and there is no fluctuation to be felt. He says that the pain which he has is all in the popliteal space.

8th.—Allowed to get up for three hours. Temperature still rises to a little over 100° at night.

15th.—Dressed after three days. About fifteen drops of discharge, which has now assumed a purulent character.

29th.—Nearly healed. Only a few drops since last dressings, four days ago. At each dressing now, knots of the catgut ligatures come away, and no doubt the slight discharge is kept up by the remains of a few of the sutures which have to come away. I can flex the knee through about 15°.

December 6th.—The wound is practically healed. During the last four days no discharge has come away from it. I find, however, small white vesicles near the anterior extremity of the scar; and upon squeezing these, small circles of un-

absorbed gut ligature came away. For the last four days he has been allowed to walk about upon crutches. The leg is supported by the old plaster-of-Paris splint. He can flex the knee through 18° or 20°, and he can lift up his leg easily with or without the splint.

20th.—He walks about well, and can flex his knee to 25° when the splint is removed.

January 10th, 1888.—No pain now, can flex the knee to 35° or 40°. There is very little lateral movement, and he can walk fairly without any splint at all on.

26th.—Leather knee-cap ordered, with steel bands behind to limit flexion. He can now bend his knee to 50° or 55°, and walks about all day.

February 9th.—Can flex the knee through 60° or 65°. A little tenderness on pressure of knee behind the inner tuberosity and the internal condyle. He can walk four or five miles with the splint on. Has the splint off at night.

On the 15th he left for Australia. I told him that he must continue to wear the knee-cap for some time, especially when riding.

On the 3rd April, 1889, he wrote from Australia to say that he was quite well. For six months he had had to be careful, but after that time the knee had given him no further trouble; and he was now able to ride unbroken colts, and to follow his usual occupation without any inconvenience.

LIST OF SPECIMENS ADDED TO THE PATHOLOGICAL MUSEUM

DURING THE YEAR 1890.

The number (62) of new specimens added to the Museum this year falls below the average of the last few years in consequence of the preparation of the new catalogue, with which fair progress has been made. It is hoped that two volumes will be in print before the end of the year.

Following our usual custom, we draw attention to those specimens which are of especial interest.

1002[56] is a specimen of spina bifida in which the tumour has disappeared, apparently from intra-uterine sloughing.

The four following are the most interesting additions to the osteological series:

1049[5] an extensive necrosis of a rib, with a large extra-pleural abscess due to pyæmia.

1097[5] a specimen of extensive caries of the scapula, probably of tubercular origin.

1117[5] a syphilitic osteitis of the ulna, which clinically presented the symptoms of new growth.

1152[87] a femur with syphilitic osteitis of twenty-five years' duration.

1501[14] is a preparation of a carotid artery which had been opened by suppuration in the middle ear, one of the rarest complications of that disease.

1519[66] and 1519[67] are illustrations of the rupture of vessels which led to the formation of false aneurysms. The patients recovered after amputation, and it was probable that the ruptures were secondary to embolisms of the vessels.

1729[21] is a typical specimen of an advanced bronchiectasis, secondary to a fibroid lung.

In 1781[41] the growth appears to be situated chiefly in the thyroid, although from its epitheliomatous structure it has evidently originated in the pharynx.

1801[11] is a specimen of multiple intestinal ulcers in an infant, the pathology of which is obscure.

In 1817[51] there is a deep pouch in the duodenum which appears to have resulted from old ulceration.

We are adding five specimens of various forms of intestinal obstruction.

In three there are evidences of old inflammatory changes which have led to kinking, fixation of a coil, and obstruction by a band respectively.

The specimen 1852, from an infant, is a congenital obstruction at the splenic flexure of the colon, a very unusual site.

1848 contains eighteen inches of the small intestine, passed per anum two years ago by a patient who still continues to enjoy good health. We are indebted to the kindness of Dr. Ewen, of Redhill, for this remarkable specimen.

1955[24] is a specimen of calcification of the gall-bladder which resulted from suppuration associated with gall-stones.

2079[70] is a rare example of miliary tuberculosis of a ureter, the infection having spread from the corresponding kidney.

Specimen 2080[76] is mounted to show an extreme degree of dilatation of the ureters in a child, for which no cause could be found. The lower part of the urinary passages was normal.

2228[97] illustrates the profuse hæmorrhage which may take place in connection with cirrhosis of the liver.

The three following specimens presented features of great clinical interest:

3034 is the larynx of a patient, about whose disease during life the greatest difference of opinion prevailed. The ulceration was variously diagnosed as syphilitic, tubercular, and lupoid.

3053b is contributed by Dr. Beaven Rake as an illustration of the association of tubercle with leprosy.

3072b. This specimen consists of the two portions of an epitheliomatous larynx successfully removed by Mr. Symonds, with great relief to the patient, who survived two years.

3128 is a thymus, weighing 10½ drachms, which was removed at the autopsy of a leucocythæmic child.

3205a is a ruptured bronchus, a lesion hitherto unrepresented in our Museum.

G. Newton Pitt.
Lauriston E. Shaw.

1002[56] Ulceration of a Spina Bifida.

The lower end of a spinal column showing an ulcerated surface an inch in diameter over the centre of the sacrum. On the reverse of the specimen a longitudinal section of the spine shows that the spinal cord terminates in the floor of the ulcer. The blue rod which projects at the upper end of the ulcer is placed in the subdural space.

From Robert M., a well-nourished infant, who died seven days after his birth. During life there was a scanty clear discharge from the ulcerated surface on the back.

Presented by Mr. L. A. Dunn, 1890.

1018 Acute Necrosis of the Atlas.

The two first cervical vertebræ showing necrosis of the whole of the posterior arch of the atlas. There is a vertical line of separation between the two portions of this arch.

Charles R., æt. 7, was admitted into the Evelina Hospital for pyrexia and a painful fluctuating swelling in the nape of the neck, which was first noticed four days before. The swelling was incised, and two ounces of pus were evacuated. The patient died of pyæmia fourteen days after admission. At the autopsy there were abscesses in the lungs, and basal and spinal meningitis. *See Lancet*, 1889, vol. i, p. 883.

Presented by Mr. Clement Lucas, 1889.

1018[16] Caries of the Atlas and Axis.

The first two cervical vertebræ, showing caries of the anterior surface of the body of the axis. The disease has extended to and destroyed the left atlanto-axial articulation. The apex of the odontoid process is roughened, and the left superior articular surface of the atlas is excavated by caries.

Joseph H., æt. 19, was admitted under Mr. Symonds for paraplegia, which came on suddenly two and a half months before. At the autopsy a prævertebral abscess was found communicating with the pharynx, and there was inflammatory softening of the spinal cord. *See Insp.*, 1890, No. 4.

1041[81] Necrosis of the Sternum.

The anterior wall of a thorax showing an abscess cavity, occupying the position of the sternum, laid open in front. Behind there is an opening which communicates with the mediastinum, and above the abscess has extended into the left sterno-clavicular joint. The body of the sternum has disappeared, and the bare ends of the costal cartilages are exposed in the abscess cavity.

Daisy B., æt. 4 weeks, was admitted into the Evelina Hospital with a fluctuating swelling over the sternum, which was opened, and from which a drachm of foul pus was evacuated. The child was the subject of inherited syphilis, and was born prematurely. It died a few days after admission to the hospital. At the autopsy pus was found in the umbilical vein. *See Lancet*, 1889, vol. ii, p. 168.

Presented by Mr. Clement Lucas, 1889.

1049[5] Pyæmic Necrosis of a Rib.

Portions of the sixth, seventh, and eighth ribs from the right side. On the outer surface of the eighth rib there is a superficial sequestrum three inches in length. Portions of the other ribs are also bare and carious. On the reverse of the specimen is an abscess cavity the size of a hen's egg, situated beneath the parietal pleura. The blue rod indicates the connection between this abscess cavity and the carious portion of the seventh rib.

Charles K., æt. 16, was admitted under Mr. Symonds for pyæmia, probably starting in connection with suppuration about the hip-joint. He died with numerous bedsores and secondary abscesses. *See Insp.*, 1888 No. 343.

1074[76] Necrosis of the Petrous Bone.

A portion of a left temporal bone, showing necrosis of the roof and posterior wall of the tympanum. The neigh-

bouring surface of bone is carious. The mastoid process has been perforated by operation.

Percy F., æt. 17, was admitted under Mr. Jacobson with double otorrhœa and symptoms of meningitis. He became hemiplegic, and the following day both mastoid processes were trephined, and some pus was evacuated from a cerebral abscess. At the autopsy there was an abscess in the left temporo-sphenoidal lobe, and pyæmic abscesses in the lungs. *See Insp.*, 1889, No. 210.

1075[72] Suppurating Gumma of the Frontal Bone.

A portion of a calvarium showing an abscess cavity situated over the right frontal eminence. There is much thickening of the adjacent pericranium, and bare bone is exposed in the floor of the abscess. The reverse of the specimen shows great thickening of the skull in the neighbourhood of the gumma.

John S., æt. 2, was admitted under Dr. Wooldridge for urgent dyspnœa requiring tracheotomy. There was a history of inherited syphilis and of a blow on the head two months before admission. At the autopsy there was extensive osteitis of the radius, membrane in the trachea, and broncho-pneumonia. *See Insp.*, 1888, No. 280; and *Prep.* 1117[10].

1097[5] Caries of the Scapula.

A portion of a left scapula removed by operation. In both the infraspinous and subscapular fossæ there is a deep irregular excavation with slightly raised edges, each measuring about two inches in diameter. On the axillary border, immediately below the roughened surface from which the head of the bone has separated, there is a third deep excavation. The spine from which the acromion process is detached is rough and carious.

William B., æt. 9, was admitted into the Shadwell Hospital for Children, for fixation of the left shoulder-joint and swelling over the scapula. There had been pain in this region for two years. Abscesses were laid open and drained over the spine and along the axillary border of the scapula, and large areæ of bare bone were exposed. Subsequently the entire bone, with the exception of its head, and the acromion and coracoid processes which had become separated, was excised. The patient slowly recovered.

Presented by Mr. L. A. Dunn, 1889.

1102[5] Osteitis of the Head of the Humerus.

A half of the head of a left humerus showing extensive condensation of the cancellous tissue. The articular cartilage is separated, and the surface of the bone beneath is carious. The epiphysial cartilage is unaffected. The fringes of synovial membrane are much thickened.

Edmund D., æt. 16, was admitted under Mr. Golding-Bird in 1887, for acute pain and swelling of the left shoulder. The head of the humerus was excised, and the joint was found to contain much purulent serum. The patient died two and a half months after the operation from general tuberculosis. *See Surgical Reports*, vol. cxxix, Case 58.

1114[31] Gunshot Fracture of the Humerus.

The upper end of a right humerus, showing on its external surface an opening leading into an irregular cavity in the head of the bone. The tuberosities are comminuted, and there are several lines of fracture crossing the articular cartilage and extending down the shaft. The margins of the opening are marked with fragments of cloth and powder.

Walter F., æt. 19, was admitted under Mr. Davies-Colley in 1890 for a severe wound of the shoulder inflicted by a shot-gun accidentally discharged a few inches in front of the patient. The head of the humerus was excised about six hours after the accident, and the patient made a good recovery. *See Surgical Reports*, 1890.

1114[81] Necrosis of the Radius.

The upper end of a right radius showing necrosis of a portion of the head and the first inch of the shaft. The sequestrum is surrounded by a thin shell of new bone.

Richard A., æt. 34, was admitted under Mr. Bryant in 1883, for acute inflammation of the elbow-joint for which his arm was subsequently amputated. *See Surgical Reports*, vol. c, Case 148.

1117[5] Syphilitic Osteitis of the Ulna.

A section of a right forearm, showing an ovoid enlargement of the shaft of the ulna, measuring an inch and a half

in diameter at its thickest part. The cut surface of the bone presents a number of small cavities which in the recent state were filled with pulpy material. The cancellous tissue is everywhere much condensed, and the periosteum is thickened. Sections of the bone examined under the microscope show the ordinary appearances of chronic osteitis without any evidence of growth or tuberculosis.

Matilda H., æt. 11, was admitted under Mr. Lucas in 1875 for a painful swelling of the right forearm which had lasted about one year. The arm was amputated, and the patient recovered. Two years later she had interstitial keratitis. The specimen was shown at the International Congress in London, 1881, as an example of "diluted syphilis," the child being the offspring of her mother's second marriage with a healthy man, her first husband having had syphilis, and the mother having at no time suffered from symptoms of the disease. *See Surgical Reports*, vol. lvi, Case 59.

1152[87] Syphilitic Osteitis of the Femur.

A section of the lower end of a left femur, showing a small abscess cavity in the centre of the medulla, which communicates by a sinus with the posterior surface of the bone. The cavity is surrounded by a zone of dense fibrous tissue, and there is considerable thickening of the compact tissue of the adjacent bone.

Henry P., æt. 45, was admitted under Dr. Taylor for lardaceous disease of the kidneys, with cerebral symptoms, from which he died. There had been sinuses in connection with the femur for twenty-five years. At the autopsy scars were found upon the penis, and there was periostitis of the tibia. *See Insp.* 1888, No. 277.

1501[9] Obliterated Carotid Artery.

Four sections of a left common carotid artery, which had been compressed by an aortic aneurysm. The origin of the vessel, which is flattened, forms the lowest section. An organised thrombus completely occludes the vessel higher up, as shown in the two middle sections, and in the upper part of the vessel it is only adherent to one side.

John S., æt. 61, was admitted under Dr. Hale White with an aneurysm of the transverse aorta, which gave rise to symptoms nine months previous to his death. *See Insp.*, 1888, No. 297.

1501[14] Perforation of the Carotid Artery, in a Carious Petrous Bone.

A left petrous bone, with the carotid artery exposed, and the middle ear opened from the front. The walls of the tympanic cavity are carious. The disease has extended above to the outer surface of the bone, and has led to ulceration and perforation of the artery at its flexure.

Wm. K., æt. 65, was admitted under Dr. Goodhart with phthisis. There had been left otorrhœa for eight years with facial paralysis for six months. Four days before his death three quarters of a pint of blood came from the left ear, the oozing continued, and some blood passed down the Eustachian tube. *See Insp.*, 1886, No. 362.

1519[66] Rupture of the Posterior Tibial Artery.

The upper part of a right leg seen from behind. The arteries have been dissected, and the pink rod indicates a rupture of the posterior tibial half an inch from its origin. There is a large cavity in the upper end of the soleus muscle, measuring three inches in length; a second cavity situated above this has extended to the front of the limb, and appears as a prominence beneath the tibialis anticus upon the reverse of the specimen. In the recent state these cavities were filled with blood-clot.

James H., æt. 32, was admitted under Mr. Lane in 1889 for gangrene of the foot and a pulsating swelling in the popliteal region. The thigh was amputated, and the patient recovered from the operation. He suffered from aortic disease, and it was supposed that the rupture was due to embolism of the artery. There were no symptoms of embolism elsewhere. *See Surgical Reports*, vol. cxliv, Case 35.

1519[67] Rupture of the Anterior Tibial Artery.

The upper part of a left leg seen from behind. The arteries are partly dissected and a rod indicates a rupture of the anterior tibial artery just at its origin. There is a large cavity five inches in length in the deep flexor muscles of the leg projecting forwards between the bones. In the recent state this cavity was filled with blood-clot. Histologically the popliteal artery is healthy.

Henry B., æt. 30, was admitted under Mr. Davies-Colley in 1889 with a non-pulsating swelling at the upper end of the calf of the left leg. There had been a compound fracture of the left tibia four years before, which healed in six months. The swelling and pain began six weeks before admission. There was a loud systolic apex murmur and albumen in the urine. The limb was amputated through the thigh, and the patient recovered from the operation. *See Surgical Reports*, vol. cxli, Case 75.

1636[80] Epithelioma of the Hand.

A right hand injected and showing a large epithelioma on its dorsal and radial surfaces. The growth measures three and a half inches in diameter, and is raised half an inch above the level of the healthy skin. Its edge is distinctly everted and its surface presents a cauliflower appearance. Histologically it is a squamous epithelioma.

William A., æt. 76, was admitted under Mr. Lane in 1890. The disease started as the result of an injury two years before admission, and had rapidly increased during the last year. The patient also suffered from a rodent ulcer of the face, which had existed for six years. *See Surgical Reports*, 1890.

1665[75] Adenoid Growths in the Naso-Pharynx of a Child.

A portion of the sphenoid and ethmoid with the vomer and turbinated bones. There are masses of adenoid tissue projecting in the middle line from the under surface of the sphenoid. On the reverse of the specimen similar smaller masses may also be seen below the ethmoid.

Joseph M., æt. 6, was admitted under Dr. Goodhart for frontal periostitis. He developed cancrum oris, from which he died. It was suggested that the adenoid growths had given rise to the periostitis. *See Insp.*, 1888, No. 109; and *Press*, 1072[66].

1729[21] Fibroid Lung with Dilatation of the Bronchial Tubes.

A section of a right lung, showing the pulmonary tissue almost entirely replaced by fibroid material, in which are seen the bronchial tubes, in some places greatly thickened, in others dilated into large intercommunicating thin-walled cavities. The pleura is much thickened.

Dennis R., æt. 13, was admitted under Dr. Taylor for severe cough, and dropsy of the lower extremities. There was a history of cough for seven years. Soon after admission fluid accumulated in the abdomen, and he was on several occasions tapped for ascites. At the autopsy the right side of the heart was found much hypertrophied; the left lung was not so severely affected as the right. *See Insp.*, 1888, No. 191.

1785[41] Epithelioma of the Pharynx Invading the Thyroid Gland.

A larynx and upper part of the trachea with a portion of the pharynx. Upon the posterior wall of the pharynx towards the left are three excavated ulcers leading to a mass of growth which has invaded the left lobe of the thyroid gland. Histologically the ulceration is epitheliomatous.

From a potman, who had suffered for two months from sore throat and loss of voice, and died rather suddenly from asphyxia. *See Note Book*, No. 3, p. 6.

Presented by Mr. Symonds, 1887.

1801[11] Simple Ulceration of the Stomach and Ileum.

A portion of a stomach, showing when held up to the light numerous minute superficial ulcers scattered over the mucous membrane. The edges are sharply defined and not thickened. The portion of ileum mounted behind shows similar ulcers of somewhat larger size. Histologically there is no evidence of tubercle.

Anne P., æt. 4 months, was admitted under Dr. Pavy for vomiting and diarrhœa. She was extremely emaciated, and died from exhaustion one week after admission. At the autopsy the other viscera were healthy. *See Insp.*, 1888, No. 358.

1817[51] A Pouch in the Duodenum.

A portion of a liver and of the duodenum. On the posterior wall of the latter is a pouch more than one and a half inches deep, with an irregular scarred surface, projecting from which are a pedunculated nodule and a bridle of fibrous tissue. The sac is surrounded by dense adipose tissue, and is adherent to the liver, which is scarred and distorted. The pouching is probably due to an old duodenal ulcer.

Charles W., æt. 39, was admitted under Dr. Goodhart for pneumonia which proved fatal next day. *See Insp.*, 1889, No. 93.

1846[20] Intestinal Obstruction, from Kinking Secondary to Contraction of the Mesentery.

A portion of the jejunum and mesentery, with enlarged non-caseous mesenteric glands. On the left of the specimen the bowel is seen to be sharply folded over at a spot where the mesentery was much shortened from old inflammation, and the lumen of the bowel is narrowed. The portion of the bowel above the obstruction lies to the right and is dilated.

Robert G., æt. 10, was admitted under Dr. Hale White for intestinal obstruction, which proved fatal in five days after the onset of the symptoms. The abdomen became distended, but was not tender. *See Insp.*, 1887, No. 303.

1848 Intussusception passed per Anum.

A foot and a half of small intestine, more or less incomplete, with ragged edges. The greater part of the mucous coat has been destroyed, leaving only the outer coats.

Ellen H., æt. 33, vomited on March 17th, 1888, and had a swelling in the right iliac fossa, which disappeared next day. On the 18th the bowels were slightly opened; the motion was fluid and tinged with blood. Opium was administered, and she was fed per rectum. On the 29th a large enema was administered, the bowels not having been opened again. The patient had emaciated greatly and was sinking. In the night three large, foul, bloody motions were passed, one of which contained the piece of bowel in the specimen, which at that time was invaginated. In May, 1890, the patient was alive and well, having had no further trouble.

Presented by Dr. Ewen, of Redhill.

1852 Congenital Occlusion of the Colon.

A portion of a colon with the rectum, showing complete occlusion of the canal at a point nine inches below the ileo-cæcal valve. Above the occlusion the intestine is much dilated, while below, though patent, it is contracted to the size of a cedar pencil. There is merely a thin septum between the two portions of the bowel at the seat of obstruction.

Albert K., æt. 7 days, was admitted under Mr. Howse for obstipation from birth. The finger could be passed into the rectum. Enterostomy was performed, and the patient died the following day. *See Insp.*, 1889, No. 129.

1852[5] Obstruction of the Cæcum by a Band.

A greatly-distended cæcum, with a portion of the liver, to the anterior margin of which is attached a band, which runs to the lateral abdominal wall, a piece of which has been preserved. Beneath this band the cæcum was obstructed, but not strangulated. The vermiform appendix (which is indicated by two blue rods) is matted down with its tips attached close to the liver. On the under surface of the cæcum are perforations due to distension ulcers, which gave way at the autopsy.

George H., æt. 62, was admitted under Dr. Pitt for intestinal obstruction. He had a fall seven days before his death. This was followed next day by abdominal pain, distension, and later on by vomiting, nothing passing per rectum. Left colotomy was performed, and he died delirious. *See Insp.*, 1887, No. 309.

1880[15] Vermiform Appendix: removed during Life.

A vermiform appendix, the muscular wall of which is greatly hypertrophied. There is no ulceration, and no retained concretion. Histologically it shows simple muscular hypertrophy.

Edward C., æt. 18, was admitted under Dr. Pye-Smith in June, 1888, for recurrent attacks of perityphlitis, which commenced in September, 1886, and for six months had been so numerous as to prevent his working. A diffused swelling in the right iliac region had been noticed for nine months. After treatment in the hospital for four months Mr. Lane removed the appendix. The patient quickly recovered, and has since then played football and taken part constantly in athletics without pain or discomfort of any kind. *See Medical Reports*, vol. cv, Case 203.

1887[68] Villous Tumour of the Rectum.

Three portions of a large sessile tumour removed by operation from the rectum. The masses are made up of closely set polypoid growths, and their surface resembles a cauliflower in appearance. In the recent state the preparation weighed three ounces.

Thomas M., æt. 73, was admitted under Mr. Symonds in 1888 for obstinate constipation and a profuse discharge of clear mucoid fluid from the rectum, which symptoms had existed for eighteen months. The mass

was felt three and a half inches from the anus. The patient was discharged from the hospital well. *See Surgical Reports*, vol. cxxxviii, Case 24.

1894[99] Malformation of the Liver.

The liver of an infant, the quadrate lobe of which is separated from the right lobe, and occupies a lower position than normal. It is attached to the longitudinal fissure by a thin pedicle of fibrous tissue, and the fundus of the gall-bladder is in contact with its posterior surface. The remaining lobes of the liver are normal.

1895[11] Atrophy of the Left Lobe of the Liver.

A portion of a liver, the left lobe of which is much shrunken, so as to measure but two inches in its vertical and horizontal diameters. This lobe has a wrinkled surface, and is very fibrous on section. Histologically the capsule is thickened, and there is considerable excess of connective tissue. The right lobe is of normal size, and shows some small depressed scars upon its surface.

Daniel M., æt. 55, was admitted under Dr. Pye-Smith with signs of phthisis. He had been ailing for some years. At the autopsy there was tuberculosis of the lungs, larynx, and intestines. The testes were fibroid. *See Insp.*, 1888, No. 389.

1955[24] Calcified Gall-bladder.

A gall-bladder which is much enlarged, so as to measure four inches in its greatest diameter. The walls are thickened, and the inner coat has undergone calcareous degeneration. There are many flocculent adhesions upon the outer surface. A gall-stone is impacted at the orifice of the hypertrophied cystic duct. Another stone, which lies at the bottom of the preparation bottle, was found in the gall-bladder together with much inspissated pus.

From a woman who was brought to the hospital dead, and in whom a dissecting aneurysm of the aorta was found at the autopsy.

2010[70] Suppurating Hydatid of the Spleen.

A spleen, weighing 24 ounces, projecting from the inner surface of which is a large cyst, with a thick fibrous wall, which has been partially cut away. The cyst contained purulent fluid, cheesy material, and numerous hydatid cysts, some of which lie at the bottom of the bottle.

Ada W., æt. 26, was admitted under Dr. Taylor with a large hepatic abscess, which was drained through the pleura. The patient died with septic pneumonia. The enlargement of the spleen was not suspected, as the colon passed in front of it. *See Insp.*, 1888, No. 381.

2079[70] Early Tuberculous Disease of a Ureter.

A portion of a bladder and ureters with small tuberculous nodules scattered over the mucous surface of the right ureter, the lower portion of which is thickened. There are superficial patches of ulceration on the inner surface of the bladder. Histologically, the nodules are tuberculous, but no bacilli were found.

Wm. W., æt. 45, was admitted under Dr. Pavy for dyspnœa and anasarca. The urine had been thick for four months. At the inspection the right kidney was found to contain tuberculous cavities, while both were affected with mixed tubal and interstitial nephritis. There were no other tuberculous lesions. *See Insp.*, 1889, No. 91.

2080[76] Dilated Ureters in a Child.

A bladder with the ureters; the latter are greatly dilated so as to measure an inch in width when laid open. The orifices of the ureters are healthy.

Alexander J., æt. 7½, was admitted under Dr. Pye-Smith for membranous laryngitis, for which tracheotomy was performed. He had been delicate from birth, but there was no history of urinary trouble. At the autopsy the pelves of the kidneys were extremely dilated; the prostate and urethra were healthy. *See Insp.*, 1886, No. 208.

2084[5] Hypertrophied Bladder.

A half of a bladder mounted to show hypertrophy of its muscular coat. The increase of thickness is much more marked in the straight anterior than in the curved posterior wall.

Samuel R., æt. 44, was admitted under Dr. Pavy for œdema of the legs and albuminuria secondary to stricture of the urethra. He died from uræmic convulsions. *See Insp.*, 1888, No. 438.

2228[97] Hæmorrhage into the Broad Ligament, associated with Hepatic Cirrhosis.

A portion of the pelvic viscera of a woman, embedded in an extensive mass of blood-clot. The bladder, which is laid open, is surrounded by a mass of clot one and a half inches thick, and there is also extravasation beneath the vesical mucous membrane.

Sarah F., æt. 38, was admitted under Dr. Pavy with cirrhosis of the liver. Extensive hæmorrhage took place into the left broad ligament, spread round the bladder and up in the abdominal wall as far as the nipple, forming a layer an inch thick. *See Insp.*, 1888, No. 359.

2289[65] Epithelioma of the Clitoris.

A portion of the vulva removed by operation from a woman, æt. 78. There is a new growth measuring one inch in diameter occupying the position of the clitoris. The growth is ulcerated, and has invaded the right nympha.

Presented by Mr. Jacobson, 1889.

2290[45] Suppuration of the Breast.

A right breast showing on the outer side of the nipple two abscesses which have burst externally. The section through the nipple shows foci of suppuration diffused through the substance of the gland. Histologically there is no evidence of malignancy or tuberculosis.

Catherine H., æt. 60, was admitted under Mr. Durham in 1888 for a swelling of the breast following a severe blow six weeks previously. The breast, which was generally enlarged, red, and œdematous, was excised, and the patient recovered. *See Surgical Reports*, vol. cxxxiii, Case 35.

2294[25] Cyst of the Breast.

A female breast seen from behind, showing an oval cyst, five inches by three inches in diameter, occupying the greater part of the gland. The posterior wall of the cyst has been

removed, exposing several papillomatous growths. The interior of the cyst is smooth, and its walls are thin and fibrous. At the lower end of the specimen there is a large part of healthy breast-tissue remaining. Histologically the papillomatous growths are composed of delicate vascular processes covered with columnar epithelium.

Presented by Mr. BRYANT, 1886.

2299[48] Adeno-Sarcoma of the Breast.

A portion of a tumour removed from the left breast. It is encapsuled, and its cut surface shows numerous minute openings, which are the sections of ducts. Histologically the growth is composed of gland-tubes and acini embedded in an abundant vascular stroma consisting of oval and spindle-shaped sarcomatous cells.

Edith M., æt. 13, was admitted under Mr. Lucas in 1879 for a swelling of the left breast, which had been noticed for one month. There was a history of a blow two years before admission. The tumour was firm, freely movable, and lobulated, and measured three inches in diameter. It was excised, and the patient recovered. *See Surgical Reports*, vol. lxxxi, Case 39.

2352[5] Cyst of the Testis.

A cyst, measuring six by three and a half inches, the wall of which is formed by the tunica albuginea of a testis. The internal surface of the cyst is rough and calcareous. At the lower and anterior part of the specimen is an oval solid mass, which represents the body of the testis. The lower end of the vas deferens, coloured red, is normal. Histologically the solid portion is chiefly composed of degenerating sarcomatous tissue.

John C., æt. 72, was admitted under Mr. Bryant for enlargement of the scrotum and a tumour of the right groin. The scrotal swelling had existed as long as the patient could remember, but the inguinal tumour was first noticed three months before admission. At the autopsy the cyst of the testis was found to contain fifteen ounces of thick, brown, mucoid fluid, which under the microscope showed cholesterine, fatty débris, and columnar ciliated epithelial cells. There was a large mass of sarcoma extending from the right iliac fossa downwards into the thigh. *See Insp.*, 1888, No. 197; and *Trans. Path. Soc.*, 1890.

2386 Epithelioma of the Scrotum.

A scrotum showing at the upper part on the right side a large oval ulcer, about two inches in its longest diameter. The edge of the ulcer is sharply defined, and its base is covered with fungating growth. By the side of the ulcer there is a prominent mass of new growth with well-marked everted edges. The adjacent skin is blackened and warty. Histologically the new growth is a squamous epithelioma.

Amos P., æt. 63, was admitted under Mr. Jacobson for swelling of, and discharge from, the scrotum. He had been a chimney-sweep for fourteen years. The growth was first noticed three months before admission, and had been ulcerated for one month. There were numerous brown warts on the inner side of the thighs. The scrotum and testes were removed, and the patient died one week later. At the autopsy early cirrhosis of the liver was found. There were no secondary deposits. *See Insp.*, 1886, No. 302.

2391[55] Destruction of the Prostate from Ulceration.

An extremely contracted bladder with the remains of the prostate and the first part of the urethra. More than half of the prostate has been destroyed, and there only remain irregular smooth bands of scar tissue, surrounding numerous intercommunicating cavities, opening into the bladder. There is no recent ulceration and no caseation. The left ureter is dilated.

James A., æt. 22, was admitted under Mr. Lucas for incontinence of urine. For five years he has had difficulty in retaining his urine, and latterly he has micturated every ten minutes. There were pus and blood in the urine. At the autopsy the right kidney weighed less than an ounce, it was fibroid, and contained small caseous nodules. The left kidney was hypertrophied and its pelvis dilated. The other viscera were healthy. *See Insp.*, 1889, No. 196.

2507[25] Intestinal Obstruction from Fixation of a Coil.

A portion of the sigmoid flexure, which is affected with malignant disease and has two fistulous communications with the ileum. The upper part of the sigmoid is dilated and slightly ulcerated. The mucous membrane of the ileum presents numerous sinuous ulcers.

Martha F., æt. 65, was admitted under Dr. Taylor for chronic intestinal obstruction. Although enemata were fairly efficient the patient became weaker, and only survived a right lumbar colotomy for a short time. At the inspection the portion of the ilium in the preparation was found fixed at the brim of the pelvis, and the intestine for two or three feet on either side were extensively ulcerated, especially at the mesenteric attachment. The ulceration was probably due to interference with the peristalsis. *See Insp.*, 1888, No. 27.

3043a Syphilitic Ulceration of the Larynx.

A larynx and upper part of the trachea, showing widespread ulceration, which extends from the base of the epiglottis to about the tenth ring of the trachea. There is little, if any, scarring, and the ulceration in the trachea presents a well-defined circular margin.

From a potman, who died suddenly while drinking in a public-house.

Presented by Dr. WITHERS GREEN, 1890.

3043b Syphilitic Ulceration of the Larynx.

A larynx laid open from the front to show deep irregular ulcers with considerable scarring and destruction of the vocal cords.

Elizabeth S., æt. 30, was admitted under Dr. Newton Pitt for aphonia and dysphagia. Six months before her admission she had been treated in the out-patient department for syphilitic ulceration of the larynx and pharynx. After remaining in the hospital four or five months she was discharged much relieved, but died rather suddenly one week later from laryngeal obstruction. *See Medical Reports*, vol. cviii, Case 153.

Presented by Dr. MAXWELL.

3053a Stenosis of the Larynx from Leprosy.

A larynx and upper part of the trachea, showing narrowing of the superior laryngeal aperture from thickening of the arytæno-epiglottidean folds and the base of the epiglottis. The rest of the epiglottis has been destroyed by ulceration.

Joseph S., æt. 21, was an inmate of the Trinidad Leper Asylum from the age of nine years. At the time of his death he had been the subject of leprosy for fourteen years. There were ulcerated tubercles on

the face, destruction of the nasal bones, and great distortion of the fingers. He died from chronic laryngeal obstruction. Numerous bacilli were found in the larynx and other organs. *See Note-Book*, No. 3, p. 7.

Presented by Dr. BEAVAN RAKE.

3053b Tuberculous Ulceration of the Larynx in a Leper.

A larynx showing severe ulceration of its internal surface with complete destruction of the vocal cords. The ulceration extends as high as the middle of the epiglottis, and in front exposes the anterior angle of the thyroid cartilage, the two halves of which have become separated.

James E., a negro, æt. 14, was admitted into the Trinidad Leper Asylum for severe laryngeal symptoms, which had first appeared a year before his admission. He died six months later from tuberculosis of the lungs. Bacilli found in the larynx were thought to be those of tuberculosis. *See Note-Book*, No. 3, p. 3.

Presented by Dr. BEAVAN RAKE.

A3061 Fixation of the Arytænoid Joints.

A larynx and trachea laid open from the front to show the inter-arytænoid space occupied by a cicatrix, which has drawn the arytænoid cartilages into close apposition, and caused fixation of their joints. There is an opening left after tracheotomy, the edges being covered with smooth skin. The interior of the larynx is healthy.

George B., æt. 32, was admitted under Mr. Durham with stricture of the urethra and ascending nephritis, from which he died. Eight years previously he was tracheotomised for syphilitic laryngitis, and had worn a tracheotomy tube continuously since the operation. *See Insp.*, 1889, No. 396.

3072b Epithelioma of the Larynx removed by Operation.

Two portions of a larynx removed at successive operations. The upper, the first removed, consists of the posterior part of the left ala of the thyroid cartilage with the true and false cords and the left arytænoid cartilage. The true cord is thickened and ulcerated, and its middle portion has been removed for histological examination. The lower portion consists of the remainder of the larynx, and shows

a large mass of new growth projecting from the right sacculus and the junction of the alæ of the thyroid cartilage. This mass depresses the anterior end of the right vocal cord. Histologically the growth is a squamous epithelioma.

From a man, æt. 41, who, after progressive aphonia for six months, was found to have an epitheliomatous growth involving the vocal cords. The whole of the projecting growth was removed by the forceps, but six months later it reappeared, and Mr. Symonds excised the left half of the larynx with the anterior part of the right vocal cord. Recurrence rapidly took place, and two months after the second operation the remainder of the larynx was extirpated. The patient recovered from the operation, and was able to speak with a fairly strong voice. He remained well for some months, but subsequently a tumour appeared in the anterior triangle of the neck, and there was some hæmoptysis. He rapidly emaciated, and died about two years after the commencement of his illness. *See Clin. Soc. Trans.*, vol. xxii, p. 245.

3076 A Cyst on the Epiglottis.

A larynx showing a thin-walled cyst, about the size of a pea, projecting from the upper free margin of the epiglottis on the right side.

3109a Enlargement of the Thyroid Gland in Graves' Disease.

A thyroid gland showing uniform enlargement. Histologically there are seen projecting into some of the numerous small cysts delicate processes covered with columnar epithelium. There is also much colloid degeneration.

Emily R., æt. 23, was admitted under Dr. Taylor with exophthalmos, enlargement of the thyroid gland, and rapid pulse. The symptoms of the disease had existed for six months. At the autopsy the thymus was found much enlarged. *See Prep.* 3129; and *Insp.*, 1889, No. 360.

A3110 Adenoma of the Thyroid Gland.

A larynx and upper part of the trachea with the thyroid gland attached. In the left lobe of the gland are two nodules, each about the size of a hazel nut, with fibrous capsules. In the fresh state the upper one was soft, and of a dark brown colour from internal hæmorrhage; the lower one was of a similar consistency, but of a pinkish-white colour. Histologically the tumours consist of groups of small spheroidal

cells arranged in follicles, among which ramify widely dilated capillaries filled with red blood-corpuscles. The adenoma is of the fœtal type.

Maria B., æt. 48, was admitted under Dr. Pye-Smith for cerebral hæmorrhage. At the autopsy there was found mitral and tricuspid stenosis, with tuberculous disease of the supra-renals and Fallopian tubes. *See Insp.*, 1889, No. 450.

3123 Cystic Adenoma of the Thyroid Gland.

An adenoma of the left lobe of the thyroid gland partially converted into a spheroidal cyst about two and a half inches in diameter.

Keziah W., æt. 41, was admitted under Mr. Symonds for left-sided enlargement of the thyroid gland. The swelling had been noticed for nine years, but had increased rapidly in the three months preceding admission. The patient made a good recovery after the removal of the cyst. *See Surgical Reports*, vol. cxliv, Case 34; and *Trans. Clin. Soc.*, 1890.

3128 Enlarged Thymus in Leucocythæmia.

A thymus gland uniformly enlarged, presenting at the upper part of the left lobe a distinct yellow discoloration, which on microscopical examination is shown to consist of an aggregation of small round cells.

Elizabeth R., æt. 2½, was admitted under Dr. Goodhart suffering from enlargement of the spleen and the lymphatic glands, with leucocytosis and anasarca. She was well until a month before admission, and died nine days later. At the autopsy the liver and spleen were found enlarged, as in splenic leuchæmia. *See Insp.*, 1889, No. 175.

3129 Enlarged Thymus in Graves' Disease.

A thymus showing uniform enlargement. Histologically there is simple hypertrophy of the gland, and Hassall's corpuscles are numerous.

Emily R., æt. 23, was admitted under Dr. Taylor with exophthalmos, enlargement of the thyroid gland, and rapid pulse. The symptoms of the disease had existed for six months. She was much exhausted by her journey to the hospital from Wales, and on admission became maniacal, refused her food, and sank in two days. *See Insp.*, 1889, No. 360.

3146a Stenosis of the Trachea following Tracheotomy.

A larynx and trachea showing an opening left after tracheotomy, below which, for the distance of about one inch, the wall of the trachea is scarred, and the lumen of the tube slightly narrowed. The larynx shows a narrow vertical scar left after median division of the thyroid cartilage.

Augustus W., æt. 2½, was tracheotomised for laryngeal obstruction. He recovered, but subsequent attempts extending over several months to remove the tube were unsuccessful. Thyrotomy was performed by Mr. Golding-Bird, and a mass obstructing the glottis removed, but it was still found impossible to dispense with the tracheotomy tube. After many attacks of bronchitis and broncho-pneumonia the child was one morning found dead in his bed. *See Insp.*, 1888, No. 409.

3166a Syphilitic Stenosis of the Trachea.

A larynx and trachea with the right and left bronchus. The trachea is thickened and narrowed throughout, and its mucous membrane is thrown into longitudinal folds. There is no appearance of ulceration or cicatrisation. The stenosis extends into the bronchi, while the larynx is quite free from any evidence of disease. Histologically there is well-marked perichondritis with fibroid thickening of the submucous tissue.

Charles C., æt. 46, was admitted under Dr. Goodhart for cough and dyspnœa. No history of syphilis was obtained. The dyspnœa became extreme, and he died cyanosed with anasarca and albuminuria. At the autopsy there was found considerable broncho-pneumonia at the base of the left lung. *See Insp.*, 1888, No. 122.

3167b Tuberculous Ulceration of the Trachea and Larynx.

A larynx and upper part of the trachea, showing two circular patches of shallow ulceration situated at the posterior attachment of the vocal cords. On the anterior wall of the trachea, at about the level of the third ring, there is a deep transverse ulcer with thickened edges, and one or two smaller superficial ulcers below it. The thyroid gland is lardaceous.

Edith B., æt. 13, was admitted under Dr. Taylor for chronic phthisis, with lardaceous disease of the liver and kidneys. There was a sinus, which had been discharging for four years, in connection with a carious rib. At the autopsy tuberculous ulceration of the intestines was also found. *See Insp.*, 1889, No. 198.

3205a Ruptured Bronchus.

The lower end of a trachea with the main divisions of the bronchi. A branch of the left bronchus is torn through at a point just below the second bifurcation of the tube.

From a woman, æt. 51, who was brought in dead, with fractured ribs and lacerated lung.

Cysticercus Cellulosæ.

Half a tongue of a pig, infected with cysticerci. Between the muscular fibres are numerous oval spaces, lined by a membrane, each containing a free hydatid.

Presented by Mr. G. Turner.

LIST

OF

GENTLEMEN EDUCATED AT GUY'S HOSPITAL

WHO HAVE PASSED THE

EXAMINATIONS OF THE SEVERAL UNIVERSITIES, COLLEGES, &c., &c.,

IN THE YEAR 1889.

University of Oxford.

First Examination for the Degree of Bachelor of Medicine.

G. H. Pennell.

Final Examination for the Degree of Bachelor of Surgery.

G. H. Pennell.

University of Cambridge.

Examination for the Degree of Doctor of Medicine.

J. E. Viney.

Examination for the Degree of Master of Surgery.

R. L. Knaggs.

Third Examination for the Degree of Bachelor of Medicine.

Part II.

A. E. Kelsey. | G. H. S. Daniell.

Part I.

G. H. S. Daniell. | E. J. D. Mitchell.
A. E. Durham. | E. M. Pilcher.
F. J. Gillibrand. | J. W. Russell.

Second Examination for the Degree of Bachelor of Medicine

Part II.

J. S. Barnes. | F. Colclough.
G. F. Birdwood. | T. H. Evans.
H. B. Bolus. | D. W. Samways.

Part I.

W. E. Alston. | D. W. Samways.
A. C. Fry. | A. Shillitoe.
A. H. Godson. | F. C. Young.

First Examination for the Degree of Bachelor of Medicine.

Part II.

C. D. Fothergill.

Part I.

H. B. Bolus. | A. Shillitoe.

Diploma in Public Health.

W. Gruggen.

University of London.

Examination for the Degree of Doctor of Medicine.

W. F. Clarke. | T. Fisher.
H. E. Crook. | C. D. Muspratt.

W. N. Risdon.

In State Medicine.

F. F. Burghard.

Examination for the Degree of Master of Surgery.

A. Parkin.

Examination for the Degree of Bachelor of Surgery.

First Division.

C. MacG. Kitching.

Obtained the Gold Medal and First Class Honours.

R. D. Mothersole.

Obtained First-Class Honours.

Second Division.

G. Black. | A. D. Fripp.

Final Examination for the Degree of Bachelor of Medicine.

May.

Second Division.

C. Price-Jones.

October.

First Division.

H. J. Campbell.

Obtained First-Class Honours in Medicine.

C. McG. Kitching.

Obtained the Gold Medal in Medicine and Honours in Obstetric Medicine.

R. D. Mothersole.

Obtained Honours in Medicine, in Obstetric Medicine, and in Forensic Medicine.

Second Division.

G. Black.
A. D. Fripp.
A. Green.
G. J. Padbury.
A. Scott.
R. E. Williams.

Intermediate Examination in Medicine.

January.

First Division.

W. G. Rogers.

Second Division.

J. W. F. Jewell. | T. G. Stevens.

Excluding Physiology.

First Division.

J. J. Browne.

Second Division.

C. B. P. Spencer. | F. W. Wilson.

July.

Honours Examination.

A. M. Daldy.

Obtained Honours in Materia Medica.

E. T. E. Hamilton.

Obtained Honours in Physiology and Histology.

T. Holmes.

Obtained Honours in Anatomy and Materia Medica.

A. T. Rake.

Obtained Honours in Anatomy, Physiology, and Histology.

Pass List.

First Division.

T. B. P. Davies.
C. C. Elliott.
P. Lord.
A. W. Sheen.
T. R. Taylor.

Second Division.

C. Braithwaite.
F. P. S. Cresswell.
E. Huntley.
T. D. Lister.
J. H. Muncaster.
H. B. Wilkinson.

Physiology only.

J. J. Browne. | C. B. P. Spencer.
F. W. Wilson.

Examination for the Degree of Bachelor of Science.

Honours Examination.

E. T. E. Hamilton.

Obtained Honours in Physiology.

T. R. Taylor.

Obtained the Scholarship in Physiology with First Class Honours.

Pass List.

First Division.

E. T. E. Hamilton.

Second Division.

T. R. Taylor.

Preliminary Scientific (M.B.) Examination.

January.

Entire Examination.

E. C. B. Ibotson.

Obtained Honours in Botany.

First Division.

H. Davies. | L. E. V. Every-Clayton.

Second Division.

R. Henderson.

One Subject only (*Chemistry*).

L. A. Parry.

July.

Entire Examination.

Honours Candidate recommended for a Pass.

H. W. Collier.

First Division.

C. E. Carpmael. | E. F. H. Hardenberg.

G. E. Manning.

Two Subjects of the Examination (*Physics and Biology*).

C. R. Colley. | L. A. Parry.
W. G. Mumford. | H. F. Turner.

One Subject of the Examination (*Physics*).

H. Harrison.

University of Durham.

Examination for the Degree of Doctor of Medicine.

W. L. Blight. | E. J. Tulk-Hart.
R. Thompson. | F. W. Turtle.

Final Examination for the Degree of Bachelor of Medicine.

R. G. P. Lansdown.

Examination for the Degree of Bachelor of Surgery.

R. G. P. Lansdown.

A. Meyrick-Jones. | F. W. Turtle.

First Examination for the Degree of Bachelor of Medicine.

J. W. H. Eyre.

In Elementary Anatomy and Physiology only.

H. Meyrick-Jones.

Naval Medical Service.

London.

W. J. Bearblock. | L. Bidwell.
W. J. Maillard.

Royal College of Physicians.

Fellowship.

P. Horrocks. | G. Newton Pitt.

Examination for the Diploma in Public Health.

J. Bampfylde. | G. F. E. Morgan.

Final Examination for the Licence.

January.

J. C. Baines.	W. Gibson.	R. A. Sawyer.
W. F. A. Clowes.	G. M. Jones	C. S. Simpson.
A. Crossley.	L. W. Keiffenheim.	W. P. Smart.
H. N. Edwards.	A. L. D. Meares.	J. W. Smith.
A. D. Fripp.	E. O. Newland.	W. G. Thistle.
C. J. Fuller.	J. Dodds-Price.	W. G. Thorpe.

April.

J. B. Booth.	E. W. Marshall.	T. F. Ricketts.
A. R. F. Evershed.	M. J. Morgan.	R. W. Rouw.
C. J. Girling.	F. J. Nisbet.	C. D. H. Rygate.
J. S. Grose.	A. E. Palmer.	T. Wilson Smith.
R. G. Hicks.	J. P. Pendlebury.	A. E. Tebb.
S. W. Mac Ilwaine.		

July.

W. H. Brenton.
H. J. Holman.
H. M. Jordan.
F. W. Pearce.
R. T. Wallace.

October.

W. G. Beyts.
G. N. P. Carrell.
W. E. Kelbe.
H. P. O. Manning.
A. E. Norburn.
P. Paget.
P. Purnell.
H. P. Rowlands.
E. E. P. Tindall.

Royal College of Surgeons.

Final Examination for the Fellowship.

H. E. Crook.
H. W. Drew.
H. Gilford.
R. W. Murray.
G. H. Pennell.
C. S. Spong.
S. Wacher.

First Examination for the Fellowship.

C. E. Pollock.
A. T. Rake.
C. E. Salter.
A. W. Sheen.
G. T. Sichel.
H. B. Wilkinson.

Final Examination for the Membership.

January.

J. C. Baines.
W. F. A. Clowes.
A. Crossley.
H. N. Edwards.
A. D. Fripp.
C. J. Fuller.
W. Gibson.
C. N. Graham.
G. M. Jones.
L. W. Keiffenheim.
A. L. D. Meares.
E. O. Newland.
R. A. Sawyer.
W. P. Smart.
A. A. Smith.
J. W. Smith.
H. Stelfox.
W. G. Thistle.
W. G. Thorpe.
T. Wilson Smith.

April.

A. R. F. Evershed.
C. J. Girling.
R. G. Hicks.
S. W. Mac Ilwaine.
M. J. Morgan.
A. G. Mossop.
F. J. Nisbet.
A. E. Palmer.
J. P. Pendlebury.
T. F. Ricketts.
R. W. Rouw.
C. D. H. Rygate.
A. E. Tebb.

July.

Bamau, das Basu.
H. J. Holman.
H. M. Jordan.
F. W. Pearce.
S. H. Seccombe.
R. T. Wallace.

October.

W. G. Beyts.
G. N. P. Carrell.
W. E. Kelbe.
H. P. O. Manning.
A. E. Norburn.
P. Paget.
P. Purnell.
E. E. P. Tindall.

MEDALLISTS AND PRIZEMEN, 1888-9.

The Treasurer's Gold Medal for Clinical Medicine.

Alfred Parkin, Hightown, Yorkshire.

The Treasurer's Gold Medal for Clinical Surgery.

Alfred Parkin, Hightown, Yorkshire.

Gurney Hoare Prize for Clinical Study.

Albert Edward Norburn, Stoke Newington.

Beaney Prize for Pathology.

Robert Devereux Mothersole, Colchester.

Sands Cox Scholarship.

Alfred Theodore Rake, Fordingbridge.
Thomas Holmes, Lancaster. *Proxime accessit.*

Michael Harris Prize for Anatomy.

Alfred Theodore Rake, Fordingbridge.

The Arthur Durham Prizes for Dissection.

First Year's Students.

John Beresford Leathes, Rochester, Prize £5.

Second and Later Years.

William Herbert Dixon, Stroud Green, Prize £15.
Arthur Mantell Daldy, Romford, Certificate.
Guy Mackeson, Fellows Road, London.

Fourth Year's Students.

Francis Griffiths Swayne, Bristol, First Prize, £25.
John Fawcett, Westgate-on-Sea, Second Prize, £10.

Third Year's Students.

John Henry Bryant, Ilminster, £17 10*s.*
Bertram Whewell Hogarth, Morecambe, £17 10*s.* } equal.

Second Year's Students.

Alfred Theodore Rake, Fordingbridge, First Prize, £25.
Thomas Holmes, Morecambe, Second Prize, £10.
Edward Thomas Ernest Hamilton, Dulwich, Certificate.
Arthur Mantell Daldy, Romford, Certificate.
Charles Caldwell Elliott, Wynberg, Cape Colony, Certificate.
Thomas Benjamin Phillip Davies, Milford Haven, Certificate.
Tom Robinson Taylor, Methley, Certificate.
Harry Bacon Wilkinson, Southport, Certificate.

First Year's Students.

Robert Henry Ritchie, New South Wales, First Prize, £50.
Frederick Gowland Hopkins, Enfield, } equal, £12 10*s.* each.
Arthur John Sharp, Bromley, Kent, }
John Beresford Leathes, Rochester, Certificate.
William John Johnson, Shefford, Certificate.

Open Scholarships in Arts.

William Sampson Handley, Loughborough, 100 Guineas.
John Robert Steinhaeuser, Brighton, 50 Guineas.

Open Scholarships in Science.

John Alexander Howard, Canonbury, 125 Guineas.
Arthur Henry Leete, Wellingborough, 50 Guineas.

Physical Society.

Session 1889–90.

Honorary President.—Samuel Wilks, M.D., LL.D., F.R.S.

Presidents.

Messrs. W. F. Colclough, B.A., A. E. Durham, B.A., J. Fawcett, J. McD. Gill, C. J. Girling, F. W. Hall, H. V. Hickman, F. G. Hopkins, C. M. Kitching, E. Moss, R. D. Mothersole, A. E. Norburn, G. B. Smith, E. H. Starling, F. G. Swayne, B.A., F. G. Vicars, C. F. Wakefield, H. W. Webber.

Pathological Committee.

Messrs. A. E. Durham, A. E. Norburn, H. V. Hickman, C. F. Wakefield.

Honorary Secretaries.

G. Newton Pitt, M.D., Louis A. Dunn, M.S.

PRIZEMEN FOR THE SESSION 1888–9.

The Treasurer's Prize for Essay on "Pyrexia" was awarded to Mr. H. W. Webber.

The Society's Prizes, First £5, was awarded to Mr. F. J. Vicars for Paper on "Puerperal Toxæmia;" Second, Mr. F. W. Hall, B.S., M.B., £2, for Paper on "Tumours of the Thyroid Body."

The Speaking Prize of £3 was awarded to Mr. J. McD. Gill, M.B.

CLINICAL APPOINTMENTS HELD IN THE YEAR 1889.

RESIDENT HOUSE PHYSICIANS.

H. E. Crook.
W. E. Tresidder.
J. L. Roberts.
A. Parkin.
E. H. Starling.
P. N. Randall.
E. Moss.

RESIDENT HOUSE SURGEONS.

A. H. Tubby.
G. Black.
E. F. Gardner.
R. D. Mothersole.
G. B. Smith.
A. L. D. Meares.

RESIDENT OBSTETRIC ATTENDANTS.

W. C. Swayne.
E. Moss.
A. Parkin.
H. E. Cuff.
G. B. Smith.
A. D. Fripp.
G. J. Padbury.
A. T. Brown.
T. Wilson-Smith.
J. P. Pendlebury.
J. E. F. Hosking.
J. W. Smith.

SURGEONS' DRESSERS.

G. H. S. Daniell.
A. Norburn.
R. A. Bremner.
J. Robertson.
T. F. Ricketts.
C. M. Kitching.
C. Price-Jones.
J. S. Richards.
F. W. Hall.
F. S. Wood.
W. J. Scott.
T. Wilson Smith.
H. A. Smith.
R. G. P. Lansdown.
J. McD. Gill.
J. W. Russell.
J. W. Roberts.
J. W. Smith.
A. E. Durham.
F. G. Swayne.
C. J. Girling.
J. Fawcett.
C. Spurrell.
S. W. Mac Ilwaine.

CLINICAL ASSISTANTS.

H. V. Hickman.
R. G. P. Lansdown.
A. D. Fripp.
J. McD. Gill.
S. W. Mac Ilwaine.
J. E. F. Hosking.
A. C. Elliman.
R. S. Freeland.
A. E. Norburn.
J. Robertson.
C. M. Kitching.
A. E. Kelsey.
R. G. Pollock.
W. J. Scott.
N. L. Richards.
G. H. S. Daniell.
H. J. Holman.
F. W. Hall.
C. H. D. Ralph.
J. W. Emmet.
J. M. Thorne.
E. M. Dobinson.
J. S. Richards.
T. S. Coombe.
N. B. Clowes.

DRESSERS IN THE EYE WARDS.

F. W. S. Davies.
W. St. Cedd.
A. L. D. Meares.
J. W. Emmet.
R. L. Kemp.
W. Fisher.
W. G. Beyts.
R. A. Sawyer.
J. M. Thorne.
F. G. Vicars.
G. M. Jones.
A. B. Hudson.
W. G. Thorpe.
H. M. Jordan.
J. E. F. Hosking.
H. V. Hickman.
A. D. Fripp.
W. E. Kelbe.
J. Robertson.
A. E. Norburn.
G. J. Padbury.
P. Paget.
W. S. Montgomery Smith.

DENTAL SURGEONS' DRESSERS.

A. L. Morris.
J. W. Smith.
W. F. A. Clowes.
F. G. Saffrey.
G. McGregor.
R. G. Pollock.
M. F. Hopson.
A. E. Kelsey.
C. J. Prime.
H. O. W. Harris.

MEDICAL WARD CLERKS.

G. F. Beresford.
E. M. Dobinson.
A. R. Lacey.
H. B. Rygate.
W. W. Wingate.
W. E. S. Cobb.
G. W. B. Featherstone.
G. H. Knapp.
W. D. Spurrell.
F. H. Brown.
W. Winslow.
E. M. Pilcher.
C. R. A. Sutton.
B. W. Hogarth.
C. H. D. Ralph.
G. S. O. Howes.
A. A. Grosvenor.
H. W. Webber.
E. G. March.
A. S. Wohlmann.
H. Hodgson.
T. G. Stevens.
W. Carling.

MEDICAL WARD CLERKS (*continued*).

W. Colclough.
W. E. Sturges-Jones.
E. H. Greaves.
C. H. D. Ralph.
A. Thomas.
J. Young.
C. G. Roberts.
H. W. John.
J. H. Bryant.
F. Wellford.
J. P. Watkins.
G. F. O'Fflahertie.
A. E. Tebb.
J. H. Roberts.
H. Richardson.
C. McGregor.
V. J. Hodgson.
C. G. Gregar.
F. G. Saffrey.
H. B. Perkins.
N. B. Clowes.
A. E. Durham.
H. A. Smith.
F. Halse-Francis.
H. B. Statter.

ASSISTANT SURGEONS' DRESSERS.

H. W. John.
F. G. Swayne.
C. Spurrell.
W. Carling.
E. H. Greaves.
F. E. Williams.
J. Fawcett.
J. H. Bryant.
N. L. Richards.
H. Richardson.
T. G. Stevens.
G. S. O. Howes.
W. K. Steele.
E. M. Rooke.
E. J. Lang.
F. D. Lumley.
E. G. March.
H. W. Webber.
A. S. Wohlmann.
A. A. Grosvenor.
F. A. Osborn.
D. W. Samways.
B. W. Hogarth.
F. H. Brown.
E. Cornish.
W. A. Higgs.
H. S. Archdall.
G. W. A. Featherstone.
A. H. Meadows.
H. E. Durham.
A. E. Norris.
G. H. Knapp.
E. M. Pilcher.
D. F. Roberts.
E. H. Baldock.
D. S. Long.
J. W. F. Jewell.
J. Young.
W. G. Rogers.
A. Thomas.
W. Winslow.
C. G. Roberts.
V. J. Hodgson.
H. L. E. Wilks.
E. W. Wheatcroft.
F. Wellford.
A. T. White.

DRESSERS IN THE SURGERY.

G. S. O. Howes.
J. Young.
A. Thomas.
E. M. Rooke.
H. W. Webber.
A. S. Wohlmann.
F. Wellford.
G. Mackeson.
E. G. March.
E. Cornish.
F. A. Osborn.
H. G. Biddle.
W. A. Higgs.
A. H. Meadows.
C. B. P. Spencer.
R. L. Wason.
F. C. Young.
H. Hodgson.
D. S. Long.
G. Pinder.
A. G. Buchanan.
W. Wilson.
E. J. H. Budge.
H. S. Archdall.
J. J. Browne.
H. L. E. Wilks.
A. E. Norris.
H. K. Rayson.
W. H. Dixon.
W. G. Rogers.
A. T. White.
E. W. Wheatcroft.
W. H. Fisher.
A. H. Godson.
E. J. Lang.
J. E. Coulson.
H. E. Durham.
T. I. Mills.
E. E. Bensley.
P. Lord.
S. J. Roberts.
A. L. Allworth.
J. R. R. Pollock.
W. A. Haslam.
T. S. Jackson.
C. E. Pollock.
V. H. Barr.
T. Holmes.
T. D. Lister.
W. S. Frith.
C. N. Thomas.

AURAL SURGEONS' DRESSERS.

C. H. D. Rygate.
H. N. Edwards.
P. Purnell.
A. E. Tebb.
G. F. O'Fflanertie.
A. T. Brown.
J. S. Richards.
F. G. Saffrey.
C. S. Spong.
A. Shilletoe.
H. V. Hickman.
J. W. Russell.

ASSISTANT PHYSICIANS' CLERKS.

V. J. Hodgson.
G. F. O'Flahertie.
J. W. Smith.
H. M. Jordan.
C. H. D. Ralph.
J. H. Roberts.
H. P. O. Manning.
D. T. Jones.
C. R. A. Sutton.
E. M. Pilcher.
G. H. Knapp.
N. L. Richards.
J. W. Emmet.
G. J. Padbury.
C. M. Dobinson.
J. Young.
J. H. Bryant.
C. G. Roberts.
H. W. John.
P. Purnell.
A. A. Grosvenor.
H. J. Van Leent.
H. Hodgson.
G. McGregor.
E. G. March.
W. D. Spurrell.
H. W. Webber.
W. E. S. Cobb.

POST-MORTEM CLERKS.

H. J. Van Leent.
R. D. Mothersole.
C. J. Girling.
J. H. Roberts.
J. Fawcett.
A. E. Durham.
W. E. S. Cobb.
C. H. D. Ralph.
E. J. D. Mitchell.
W. Wingate.
C. J. Prime.
H. W. John.

OBSTETRIC WARD CLERKS.

A. B. Hudson.
F. W. Pearce.
H. Cox.
F. W. Welstead.
C. J. Fuller.
A. T. McConkey.
J. Cruickshank.
E. M. Dobinson.
A. E. Tebb.
F. W. A. Clowes.
W. C. Burt.
H. Richardson.
A. E. Kelsey.

OBSTETRIC OUT-PATIENTS' CLERKS.

J. W. Russell.
C. H. D. Ralph.
D. Rubinstein.
F. W. Welstead.
G. F. O'Flahertie.
W. Carling.
W. S. Montgomery-Smith.
F. G. Swayne.
S. W. MacSwaine.
F. D. Lumley.
C. M. Kitching.
J. S. Richards.
F. W. A. Clowes.
W. E. S. Cobb.
E. M. Pilcher.
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1890.

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S. Wilks, M.D., LL.D., F.R.S.
F. W. Pavy, M.D., LL.D., F.R.S.

Consulting Obstetric Physicians.

H. Oldham, M.D.
J. Braxton Hicks, M.D., F.R.S.

Physicians.

P. H. Pye-Smith, M.D., F.R.S.
Frederick Taylor, M.D.
J. F. Goodhart, M.D.
W. Hale White, M.D.

Assistant Physicians.

G. Newton Pitt, M.D.
E. C. Perry, M.D.
L. E. Shaw, M.D.
J. W. Washbourn, M.D.

Obstetric Physician.

A. L. Galabin, M.D.

Assistant Obstetric Physician.

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Charters J. Symonds, M.S.
W. Arbuthnot Lane, M.S.

Ophthalmic Surgeon.

C. Higgens, Esq.

Assistant Ophthalmic Surgeon.

W. A. Brailey, Esq.

Aural Surgeon.

W. Laidlaw Purves, Esq.

Instructor in Anæsthetics.

Tom Bird, Esq.

Surgical Registrar.

A. Parkin, M.S.

Curator of the Museum.

Dr. Shaw.

Lying-in Charity.

Dr. Galabin and Dr. Horrocks.

Dean.

Dr. Perry.

LECTURERS AND DEMONSTRATORS.

Clinical Medicine	The Physicians and Assistant Physicians.
Clinical Surgery	The Surgeons and Assistant Surgeons.
Medicine...	Dr. Pye-Smith and Dr. Taylor.
Practical Medicine	Dr. Goodall.
Surgery	Mr. Howse and Mr. Davies-Colley.
Practical Surgery...	Mr. Symonds and Mr. Lane.
Operative Surgery	Mr. Symonds.
Midwifery and Diseases of Women	Dr. Galabin.
Practical Obstetrics	Dr. Horrocks.
Mental Diseases	Dr. Savage.
Ophthalmic Surgery	Mr. Higgens and Mr. Brailey.
Dental Surgery	Mr. Newland-Pedley.
Aural Surgery	Mr. Laidlaw Purves.
Diseases of the Skin	Dr. Perry.
Diseases of the Throat	Mr. Symonds.
Electro-Therapeutics	Dr. Washbourn.
Anæsthetics	Mr. Tom Bird.
Hygiene and Public Health... ...	Mr. Turner.
Pathology	Dr. Pitt.
Morbid Anatomy	Dr. Pitt, Dr. Perry, and Dr. Shaw.
Morbid Histology	Dr. Pitt and Mr. Lane.
Bacteriology	Dr. Washbourn.
Medical Jurisprudence...	Dr. Stevenson.
Anatomy...	Mr. Lucas and Mr. Jacobson.
Practical Anatomy	Mr. Dunn, Mr. Targett, and Mr. Fripp.
Physiology	Mr. Golding-Bird.
Histology and Practical Physiology	Mr. Starling and Mr. Tubby.
Materia Medica and Therapeutics	Dr. Hale White.
Practical Pharmacy	The Hospital Dispenser.
Chemistry	Dr. Stevenson.
Practical Chemistry	Mr. Groves.
Experimental Physics	Professor Reinold.
Biology	Mr. Beddard and Mr. Campbell.

The Hospital contains 695 beds, of which 500 are in constant occupation. Special Classes are held for Students preparing for the Examinations of the University of London, and other Higher Examinations.

APPOINTMENTS.

All Hospital Appointments are made strictly in accordance with the merits of the Candidates, and without extra payment.

ENTRANCE SCHOLARSHIPS.

The following Entrance Scholarships are awarded.

Two Open Scholarships in Arts, one of the value of 100 guineas open to candidates under 20 years of age; and one of 50 guineas open to candidates under 25 years of age. Two Open Scholarships in Science, one of the value of 125 guineas, and another of 50 guineas, open to candidates under 25 years of age.

PRIZES

Are awarded to Students in their various years, amounting in the aggregate to more than £330.

DENTAL SCHOOL.

A Dental School is attached to the Hospital, which affords to Students all the instruction required for a Licence in Dental Surgery.

COLLEGE.

A Residential College has been erected upon a site close to the Hospital, to accommodate about 50 Students in addition to the Resident Staff of the Hospital. The College contains a large Dining Hall, a Smoking Room, a Gymnasium, and Reading Rooms for the use of the STUDENTS' CLUB.

For Prospectus and further information apply to the Dean, Dr. PERRY, Guy's Hospital, London, S.E.

GUY'S HOSPITAL.

DENTAL SCHOOL.

Attendance of Assistant Dental Surgeons and Anæsthetists.

Monday.

Mr. H. Pillin, L.D.S.E. . . . . Dr. Sheppard.

Tuesday.

Mr. Mansbridge, L.R.C.P., M.R.C.S., L.D.S.E. Dr. Silk.

Wednesday.

Mr. Richards, M.R.C.S., L.D.S.E. . . . Mr. Lloyd.

Thursday.

Mr. Maggs, L.R.C.P., M.R.C.S., L.D.S.E. . . Dr. Silk.

Friday.

Mr. Rouw, L.R.C.P., M.R.C.S., L.D.S.E. . . Dr. Sheppard.

Saturday.

Mr. Murray, L.D.S.E. . . . . Dr. Cock.

The *Dental Surgeon*, Mr. Newland-Pedley, F.R.C.S., L.D.S.Eng., attends on *Tuesdays* in the Extraction Room to see special cases.

LECTURES AND DEMONSTRATIONS.

Winter Session.

Dental Surgery . . . . Mr. Newland-Pedley.
Dental Anatomy and Physiology Mr. Maggs.
Metallurgy . . . . . Mr. Groves.

Summer Session.

Dental Mechanics . . . Mr. Richards.
Operative Dental Surgery . . Mr. Murray.
Dental Microscopy . . . Mr. Mansbridge.
Dental Tutor . . . . Mr. Rouw.

The Special Lectures are given at 4 p.m.

The Conservation Room,* which has been recently built for the special use of the Dental School, is brilliantly lighted by six windows in front, and also from a glass roof. It affords accommodation for eighteen Dental Chairs on three tiers, and is open from 9 a.m. to 3 p.m. for the performance of all the operations of conservative dentistry.

A Clinical Demonstration is given at 11 a.m. by the Assistant Dental Surgeon for the day on one of the various methods of filling, the preparation of roots for pivots, and the adaptation of porcelain and metal crowns to broken-down teeth. Electric Motors and Mallets are employed in these demonstrations, and a Dynamo has been installed.

There is an **Extraction Room*** for ordinary cases, and a **Gas Room*** for operations under Anæsthetics, which are administered daily to such patients as the dental officers recommend. Cases suitable for conservative treatment are transferred to the Conservation Room at the discretion of the Assistant Dental Surgeons.

The Dental Laboratory* is more than 23ft. long, and lighted by five windows in front, and by three small skylights over the Casting Bench, the Lathe, and the Plaster Bench. There are seats for twelve students, and they are allowed to construct and fit dentures and correction-plates for patients, thus gaining valuable experience in taking models, making "bites," and adjusting artificial teeth in the mouth. Suitable cases are found in the Hospital, and the work done is absolutely free of charge. Practical instruction is given to supplement the Course of Lectures on Dental Mechanics, and Demonstrations are held on the construction of dental splints, obturators, and vela, used in treating patients in the wards, where there are always interesting cases of surgical disorders, complicated with dental lesions.

The Tutor holds classes before each Examination to assist Candidates in preparing for the L.D.S.

The Fee for the General and Special Lectures and Hospital Practice required for the L.D.S.Eng. is £70 on entrance, or 70 guineas by instalments. The inclusive Fee for L.R.C.P., M.R.C.S., L.D.S.Eng. is £150 on entrance, or 150 guineas by instalments. Further information with Prospectus can be obtained from the Dean, Guy's Hospital, S.E.

* The whole of the Dental Department is being enlarged to three times its present size.

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And it is expressly declared in the Will "That no Physician or "Surgeon, or other officer for the time being, of Guy's Hospital or of "St. Thomas's Hospital, in the Borough of Southwark, nor any person "related by blood or affinity to any such Physician or Surgeon, for "the time being, or to any other Officer for the time being in either "of the said Hospitals, shall at any time receive or be entitled to "claim the Prize." But, with the exceptions here referred to, this Prize is open for competition to the whole world.*

Candidates are informed that their Essays, either written in the English Language, or, if in a Foreign Language, accompanied by an English translation, must be sent to Guy's Hospital on or before January 1st, 1892, addressed to the Physicians and Surgeons of Guy's Hospital.

Each Essay or Treatise must be distinguished by a Motto, and be accompanied by a sealed envelope containing the name and address of the Writer. None of the envelopes will be opened except that which accompanies the successful Treatise. The unsuccessful Essays or Treatises, with the illustrative preparations or drawings, will remain at the Museum of Guy's Hospital until claimed by the respective writers or their agents.

* The Prize cannot be awarded to any Essay which is the joint production of two or more authors.

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CONTENTS.

1. In Memoriam:—George Owen Rees, M.D., F.R.S., Leonard Charles Wooldridge, M.D., Samuel Osborne Habershon, M.D.
2. Two Cases of Division of the Musculo-spiral Nerve in which Resection and Suture were followed by Complete Restoration of Function. By R. Clement Lucas, B.S.
3. The Pathology of the Human Sympathetic System of Nerves. By W. Hale White, M.D.
4. A Case of Cystic Bronchocele, with a Synopsis of Cases of Bronchocele treated in Guy's Hospital from 1877 to 1888 inclusive. Being a portion of a Paper which won the Gurney Hoare Prize. By G. Herbert Pennell. (Communicated by the Editors.)
5. On the Etiology and Clinical Aspects of Scarlatinal Nephritis. By E. W. Goodall, M.D.
6. A Case of Complete Primary Absence of the Corpus Callosum. By L. A. Dunn, M.S.
7. On the Evolution of Mental Disorder. (Insane Self-consciousness, Doubt, and Jealousy.) By G. H. Savage, M.D.
8. On the Treatment of Acute Pneumonia, more particularly by the Local Application of Ice to the Chest. By James F. Goodhart, M.D.
9. Necrosis of the Coccyx. By L. A. Dunn, M.S.
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11. Deltoid Neuralgia. By C. H. Golding-Bird.
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13. The Pre-Cancerous Stage of Cancer of the Tongue. By W. H. A. Jacobson, M.Ch.
14. Traumatic Separation of the Epiphyses of the Upper Extremity. By A. H. Tubby, B.S.
15. The Maybrick Trial and Arsenical Poisoning. By Thomas Stevenson, M.D.
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19. A Case of Simultaneous Disiocation of both Ends of the Clavicle. With Remarks on ten previously reported Cases. By R. Clement Lucas, B.S.
20. A Short account of the Residential College, and of some of the Changes recently made in the Resident Appointments of the Medical School of Guy's Hospital. By E. C. Perry, M.D.
21. List of Specimens added to the Pathological Museum during the years 1888–89. By G. Newton Pitt, M.D., and J. H. Targett, M.S.

List of Pupils who have passed the Examinations of the several Universities, Colleges, &c., 1888.

List of Prizemen and Medallists, 1887–88.

List of Pupils who have received appointments at Guy's Hospital, 1888.

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